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VISUAL DEFECTS CAUSED BY OCCIPITAL LOBE LESIONS

REPORT OF THIRTEEN CASES *

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Unusual opportunities are afforded by war injuries for the observation of disturbances of the visual functions in their relation to more or less circumscribed lesions of the brain, and several important contributions to this subject have been published in foreign journals since the beginning of the world war. In 1916, Holmes and Lister [1] reported their observations on a large series of cases of this type and deduced the following conclusions:

1. The upper and lower halves of each retina are represented in the upper and lower halves of each visual area, respectively.

2. The center for macular vision lies in the posterior part of the visual area, probably on the margins and lateral surfaces of each occipital pole. The macula has not a bilateral representation.

3. The center for vision subserved by the periphery of the retina is situated in the anterior part of the visual area, and serial concentric zones of the retina from the macula to the periphery are represented in this order, from behind forward in the visual cortex.

4. Those parts of the retinas adjoining their vertical axes are probably represented in the dorsal and ventral margins of the visual areas along the mesial surfaces of the occipital lobe, while the retina in the neighborhood of its horizontal axis is projected onto the walls and floor of the calcarine fissures.

5. Severe lesions of the visual cortex produce complete blindness in corresponding parts of the visual fields or, if incomplete, an

* Read at the Forty-Eighth Annual Meeting of the American Neurological Association, May, 1922, Washington, D. C.

1. Holmes and Lister: Disturbance of Vision from Cerebral Lesions, Brain **39**:34, 1916.

amblyopia, color vision being generally lost and white objects appearing indistinct, or only more potent stimuli, as abruptly moving objects, may excite sensations.

6. Defects of vision in the two eyes are always congruous and superimposable, provided no abnormalities of the peripheral visual apparatus exist.

7. Lesions of the lateral surface of the hemispheres, especially of the posterior parietal regions, may cause certain disturbances of the higher perceptual functions with intact visual orientation, and localization in space; disturbance of the perception of depth and distance; loss of visual attention and agnosia.

Again in 1918, Holmes [2] called attention to the various types of hemianopsias, quadrant defects, multiple, paracentral and central scotomas that resulted, depending on the location of the lesion and the amount of tissue destroyed. Visual charts were presented describing these defects, with explanations as to the probable site of the lesion. In a few instances postmortem findings were also given.

In the same year, Holmes [3] contributed an article on the "Disturbance of Visual Orientation," grouping the symptoms of the condition under two main headings: (*a*) the disturbance of orientation and localization in space by sight; (*b*) the disturbance of movement of the eyes and ocular reflexes.

In 1917 Riddock [4] published a paper on dissociations of visual perceptions due to occipital injuries and referred especially to the appreciation of movement in the blind field and its prognostic value, and to the types of dissociation being analogous to the dissociation of general somatic sense impressions as occurring in cerebral injuries. He corroborated the findings of Lister and Holmes in regard to cerebral localization of the macula, etc.

A year later Moreau,[5] studying occipital injuries from a slightly different angle, divided the retina from the periphery centrally into three zones of perception, distinction and fixation. He said that it was necessary to study from a physiologic standpoint the reason why central vision was so often intact in cases of lateral and even bilateral hemianopsia, as publications had been too much concerned with the lost areas of peripheral vision. He thought complementary studies of central vision were more important, especially the physiologic and

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2. Holmes: Disturbances of Vision by Cerebral Lesions, Brit. J. Ophth. **2**:353, 1918.

3. Holmes: Disturbance of Visual Orientation, Brit. M. J. **2**:449, 506, 1918.

4. Riddock: Dissociations of Visual Perceptions Due to Occipital Injuries, with Especial Reference to Appreciation of Movement, Brain **40**:15, 1917.

5. Moreau: Sur les troubles de la vision maculaire produit par les lesions traumatique de la region occipitale, Ann. D'Oculistique **155**:357, 1918.

auto-examinations; and that these should precede the clinical, as the physiologic point of view is important in discussing the independent existence of foveal and macular fibers, whether crossed, direct or mixed.

Morax [6] said he had never found fixation affected in hemianopic scotomas due to a unilateral occipital lesion, but he had found it lost when both occipital lobes were affected. He made a distinction between the periphery of the macular area and the fixation point.

Referring to a previous article by Morax, Moreau and Castelain,[7] he said that they had found the same alterations in the periphery of the macula as in the peripheral field properly speaking; that is, either a quadrant, complete or irregular hemianopsia. This may occur either in conjunction with the peripheral field alterations or not, thus presupposing two centers, one for the macula and one for the periphery.

Concerning the state of vision at the point of fixation in cases of macular and peripheral hemianopsias or scotomas, they never found it altered. Morax says it is necessary to admit of a small zone at the point of fixation, of probably 1 degree in extent, which possesses very complex connections. His conclusions are practically the same as those of Holmes, but he states more clearly that the preservation of normal acuity of vision is compatible with the destruction of one macular center, that is, either the right or left occipital lobe.

Wilbrand and Sanger [8] evidently accept the view that the macula, or at least the center of it, has bilateral cortical connections. They cite cases, however, in which hemianopic fields are blind to the fixation point, without loss of acuity of central vision.

The patients forming the basis of this report were under observation in General Hospital No. 11 at Cape May in 1919, and in each case had received a wound of the head which involved one or both occipital lobes. In each of three cases (1, 5 and 7) a foreign body had traversed the brain for a considerable distance, in two instances (1 and 7) passing from one hemisphere to the other across the median line. In Cases 5 and 7 the roentgen ray revealed projectiles in the substance of the brain near the opposite side of the cranium from the points of entrance. In Case 1 there was evidence that a projectile had been removed by early operation at a distance from the wound of entry. In several cases small fragments of bone were revealed within the cranial cavity near the defect in the skull.

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6. Morax: Discussion des hypothesis faites sur les connexions corticales des faisceau maculaires, Ann. D'Oculistique **156**:25, 1919.

7. Morax, Moreau and Castelain: Les differents types d'alterations de la vision maculair dans les lesions traumatique occipital, Ann. D'Oculistique **156**: 1, 1919.

8. Wilbrand and Sanger: Neurologie des Auges, 1917.

While none of the patients in this series came to necropsy and it was impossible to determine with accuracy the extent of the brain injuries, it is of some interest to observe the correlation of the visual defects and the brain lesions.

In charting the fields a self registering perimeter was used to determine the peripheral fields and an improvised screen similar to the Bjerrum screen for the purpose of mapping more accurately the visual defects of the central areas. Using an object 5 mm. square, and with the patient at a distance of 1 meter from the screen, defects in the central areas may be recorded with a margin of error of less than 1 degree.

The illustrations showing the position of cranial defects and of foreign bodies are based on tracings from radiographs. Conventional diagrams of the cranium, that of the lateral aspect modified from Marie and Foix,[9] and of the posterior aspect from Wilbrand and Sanger,[8] are utilized, depicting the bony landmarks in relation to those of the cerebral cortex. The close approximation of the posterior poles of the occipital lobes to the external occipital protuberance is particularly to be noted.

REPORT OF CASES

Case 1.—P. B., 26 years of age, was wounded Sept. 27, 1918, in the left occipital region, after which he was unconscious for several hours. There was an early operation the details of which are unknown. Following this he was paralyzed in the left arm and leg for two or three months. He was admitted to Hospital No. 11, March 13, 1919. Roentgen-ray examination revealed a cranial defect in the left occipitoparietal region (Fig. 1), a small metallic fragment near the midline intracranially, trephine holes and the outlines of a bone flap in the right parietal bone. There was a trace of a residual left hemiplegia and a right homonymous hemianopsia. Eye movements, pupils and fundi were normal. Fixation was retained. Visual acuity: right eye 20/15, left eye 20/20. The field charts (Fig. 1) show the blind areas extending to within less than 1 degree of the fixation point, and about the same distance from the midlines in the upper halves of the fields. In this case evidence justifies the conclusion that a projectile entered the left occipital region at the site of the cranial defect, passing upward, forward and to the right, and lodged in the right parietal region from whence it was removed by an early operation.

Case 2.—W. C., a man aged 25, had been wounded Sept. 29, 1918, by a shell fragment in the left posterior parietal region near the midline. He was unconscious for about five minutes, and there is no record of an early operation. He was admitted to Hospital No. 11, March 10, 1919, with an unhealed wound. Roentgen-ray examination showed a cranial defect 2 cm. in diameter near the posterior superior angle of the left parietal bone (Fig. 2), also numerous small bone fragments near the margins of the defect and a small metallic foreign body 2 cm. downward from the inner table. Homonymous hemianopsia constituted the only focal symptom of cerebral injury. Ocular movements, pupils

9. Marie, Pierre and Foix, C.: Les aphasies de guerre, Rev. neurol., February, 1917.

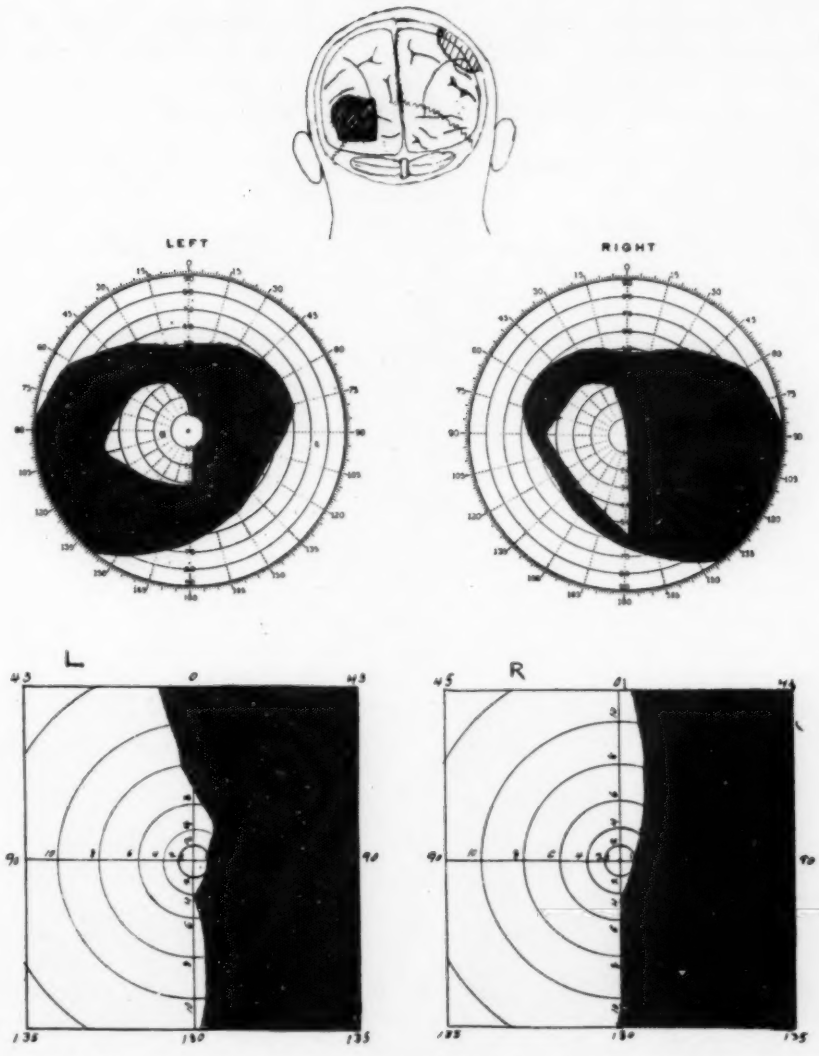

Fig. 1 (Case 1, P. B.).—Left parieto-occipital wound and cranial defect; right parietal bone flap; right homonymous hemianopsia. Perimeter charts.

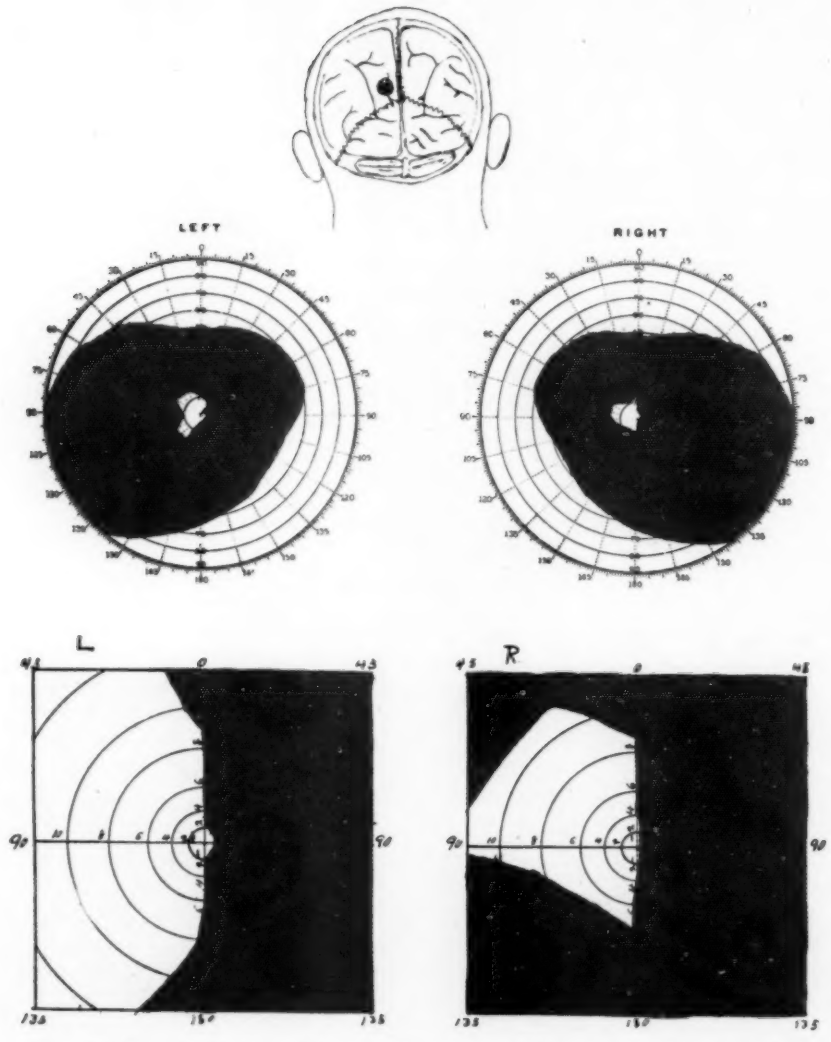

Fig. 2 (Case 2, W. C.).—Wound and cranial defect in left posterior superior parietal region; incomplete right homonymous hemianopsia. Perimeter charts.

and fundi were normal, and fixation was retained. Visual acuity: right eye 20/20, left eye 20/20. Field charts made with a 5 mm. test object showed homonymous hemianopsia extending to within 0.5 degree of fixation points. Tests made with bright lights and large moving objects revealed a slight degree of retained vision throughout the affected fields. From the location of the cranial defect and the relative position of the metallic and bone fragments, it is apparent that the cerebral lesion involved the left occipital lobe near the upper margin at a considerable distance from the occipital pole. The destructive effect of the wound was evidently downward, toward the optic radiations of Gratiolet.

CASE 3.—F. C. B., aged 23, was wounded Nov. 1, 1918, in the right occipital region, after which he was unconscious for one half hour. There was no record of an early operation. The wound healed in February, 1919, and he was admitted to Hospital No. 11 a month later. A roentgenogram proved that there was a cranial defect 5 cm. in diameter involving the right occipitoparietal suture (Fig. 3). There were numerous small bone fragments in the vicinity of the defect. A left homonymous hemianopsia was the only focal cerebral symptom observed. Ocular movements and pupils were normal; both fundi showed slightly blurred disk margins. Visual acuity: right eye 20/20, left eye 20/20. Fixation was retained. The charts show hemianopic fields extending to within 0.25 degree of the fixation points (Fig. 3).

CASE 4.—J. B., aged 24 years, received multiple wounds in the shoulder and back from a bursting shell on June 13, 1918, and was wounded a second time on Nov. 3, 1918, a piece of metal passing through the right orbit and lodging in the right temporal muscle without penetrating the cranium. The right eye was enucleated, but there was no record of an occipital wound. This patient was admitted to Hospital No. 11, Feb. 20, 1919. Roentgen-ray examination showed: (*a*) a metallic foreign body, 1.5 cm. in diameter, in the right temporal muscle above the zygoma; (*b*) a cranial defect 2 cm. in diameter in the occipital bone 1 cm. to the right and 1 cm. above the external occipital protuberance. The field chart shows a left hemianopsia extending practically to the fixation point. The visual acuity was 20/30. Although the patient was unaware of the occipital wound, and the history contained no record of it, circumstances indicate that it occurred at the time he received the wound on the back and shoulder. If that surmise is correct, this soldier returned to duty with an unrecognized homonymous hemianopsia.

CASE 5.—G. C., aged 31 years, a man, received a wound Nov. 3, 1918, in the left occipital region near the external occipital protuberance. He was transferred to Hospital No. 11, Dec. 28, 1918, for observation. On admission the following conditions were present: marked right hemiplegia, complete aphasia and right homonymous hemianopsia. The hemiplegia gradually receded to a slight residual remnant. Speech and the comprehension of spoken language were in large measure regained; but almost complete alexia persisted. The hemianopsia, however, was complete and permanent. Roentgen-ray findings were: (*a*) a cranial defect, 1 by 0.75 cm., 1 cm. above the inion, bordering the midline; (*b*) a metallic foreign body in the frontal region close to the midline, about 3 cm. under the coronal suture (Fig. 5). Ocular movements and pupils were normal, and the fundi were negative with the exception that the left disk margin was blurred. Fixation was retained and visual acuity was: right eye 20/20, left eye 20/20. Charts of the macular areas show the blind fields extending to within 0.66 of a degree of the fixation point.

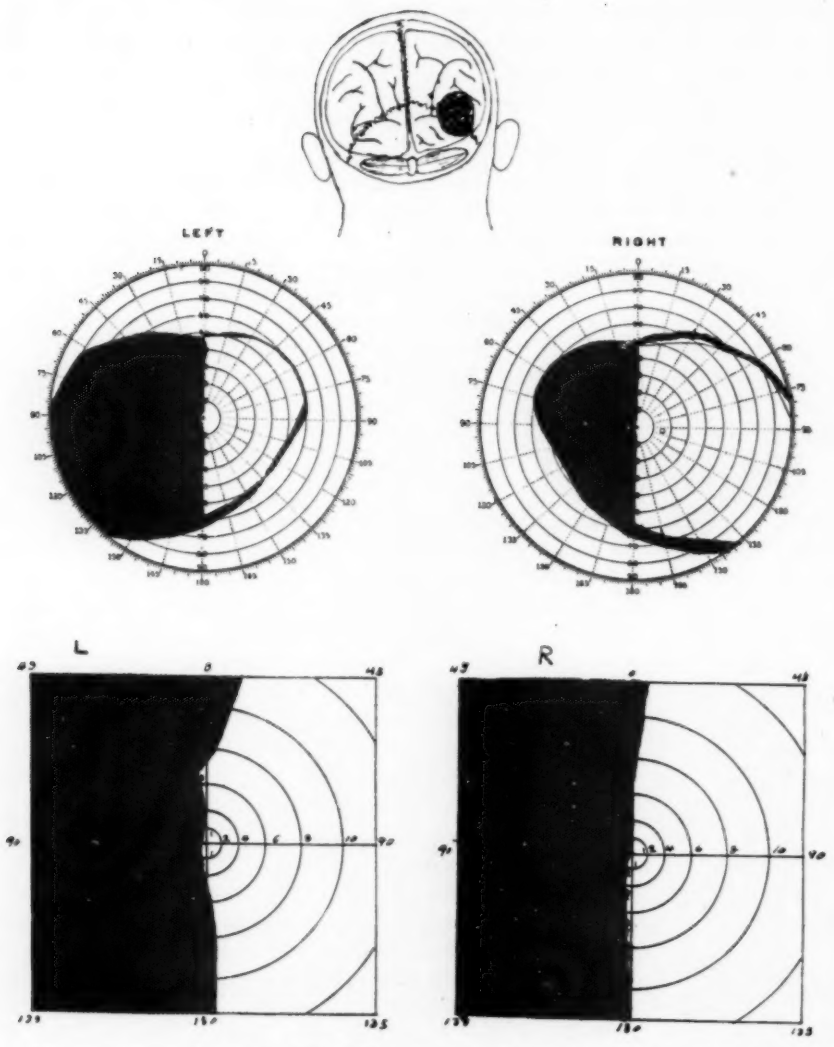

Fig. 3 (Case 3, F. C. B.).—Wound and defect in right parieto-occipital region; left hemianopsia. Perimeter charts.

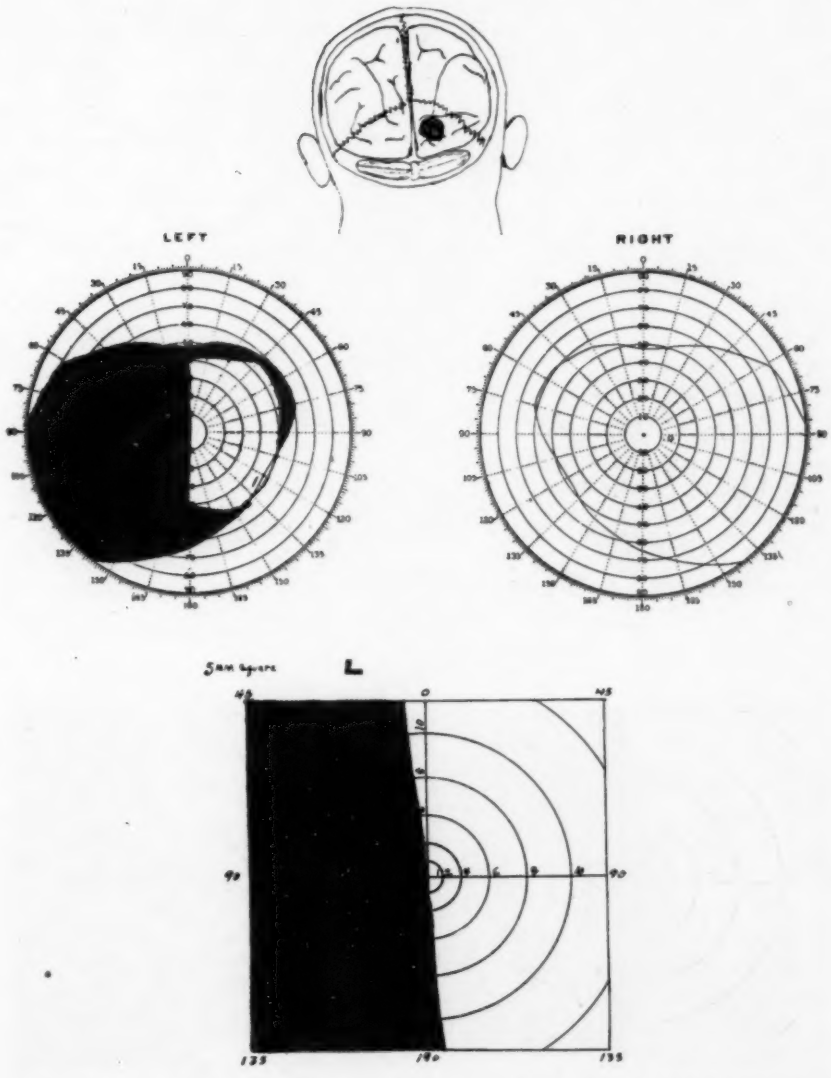

Fig. 4 (Case 4, J. B.).—Right occipital wound and defect; left hemianopsia. Perimeter charts.

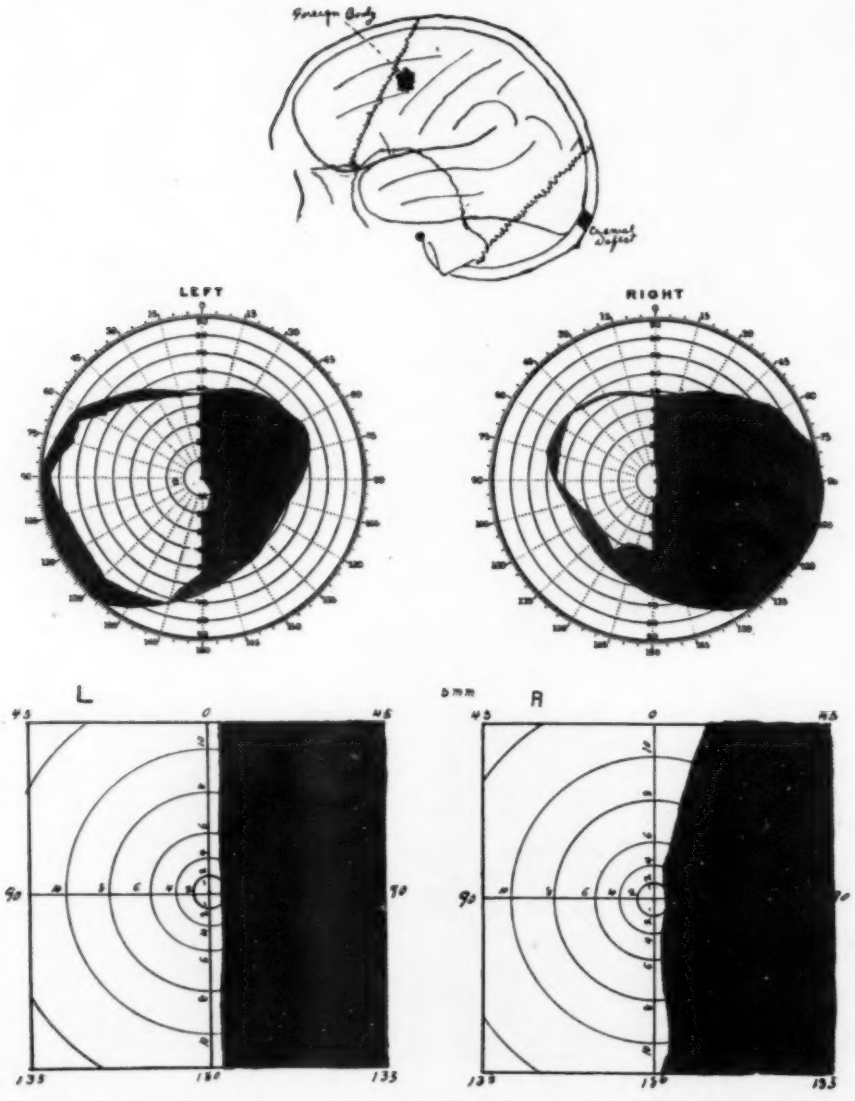

Fig. 5 (Case 5, G. C.).—Left occipital wound and defect near inion. Metallic foreign body in frontal region close to midline; right hemianopsia, alexia; transient right hemiplegia and complete aphasia. Perimeter charts.

Case 6.—No details of the early history of A. D., aged 25 years, were available. A roentgenogram revealed a large irregular cranial defect, 4 by 8 cm., involving the left occipital and parietal bones, and bordering on the midline (Fig. 6). The ocular movements, pupils and fundi were normal, and slight haziness of the lens of the right eye was present. Visual acuity was: right eye 20/100, left eye 20/15. A right homonymous hemianopsia extended to the fixation point (Fig. 6).

Case 7.—Early records of M. D., aged 24, were not available, and he was unable to give details on account of memory defect. He was ambulatory with evidences of a slight residual left hemiparesis, mentally confused and amnesic and gross intelligence defect was present. He had difficulty at first in finding his bed, and was frequently lost in the corridors of the hospital. Roentgen-ray findings were: (*a*) cranial defect 3 cm. in diameter in the right frontal region; (*b*) metallic foreign body about the size and shape of a machine-gun bullet in the left occipital region 2 cm. from the inner plate of the occipital bone, 3 cm. to the left of the midline and 1 cm. above the level of the inion. Ocular movements, pupils and fundi were normal. Visual acuity was: right eye 20/50, left eye 20/50. There was a left homonymous hemianopsia present extending to within 5 degrees of the fixation point. From the roentgen-ray evidence the foreign body was apparently lodged just posterior to the posterior horn of the left lateral ventricle. Its probable course may be assumed to extend from the frontal defect downward, backward and to the left across the midline. The right corona radiata was traversed, as was the posterior part of the corpus callosum. The optic radiations of the left occipital lobe were also somewhat involved.

Case 8.—F. D., 26 years, was wounded Oct. 28, 1918, in the right occipital region. He was unconscious for ten hours, and on waking "everything seemed blurred." In an early operation, under local anesthesia, bone fragments and pulped brain tissue were removed, but no foreign body was found. He was admitted to Hospital No. 11, April 3, 1919, ambulatory. A roentgenogram showed a cranial defect 3 by 4 cm. in occipital bone bordering the right lambdoidal suture and several small bone fragments down and in from the defect. The ocular movements were normal, and the pupils were equal. The reflexes were present, the left being a little sluggish; vessels of both fundi were tortuous, disks sharply defined; the physiologic cup was obliterated on the right and small on the left. Visual acuity: right eye 20/15, left eye 20/15. Left homonymous hemianopsia extended to the fixation points.

Case 9.—L. S., a man aged 29, was wounded Oct. 12, 1918, in the left occipital region. An early operation having been performed, he was admitted to Hospital No. 11, Jan. 4, 1919, remaining under observation for six months. A roentgenogram showed a defect 5 by 6 cm. in the left occipital region bordering the midline. An operation for the repair of the cranial defect was performed, March 15, 1919. A cone-shaped, fluid filled cavity was encountered, the apex of which communicated with the posterior horn of the left lateral ventricle, the base coinciding with the margins of the cranial defect. A considerable quantity of cerebrospinal fluid escaped, and the roentgenogram taken immediately after the operation showed the outline of the air-filled lateral ventricle (Fig. 9). The ocular movements were normal; however, slight convergence of the right eye was present. The pupils were equal, reflexes present, media clear, and there were no significant fundus changes. Visual acuity was: right eye 20/50, left eye 20/30. There was a complete right homonymous hemi-

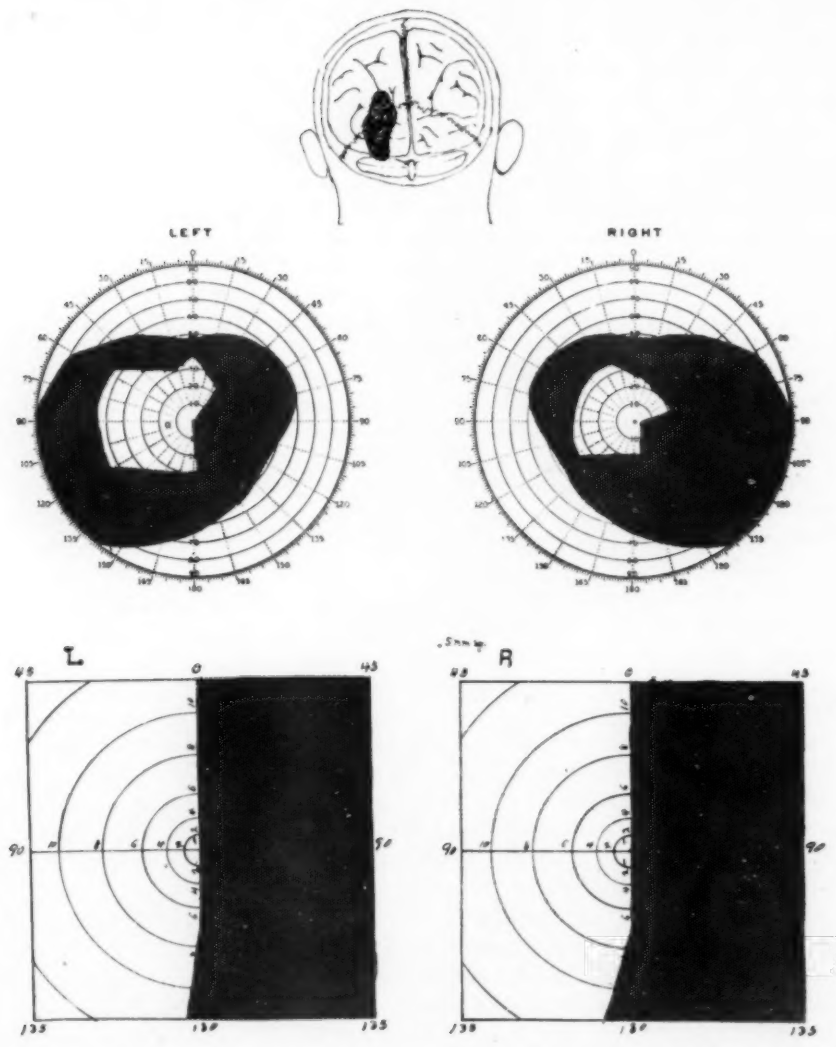

Fig. 6 (Case 6, A. D.).—Left parieto-occipital wound and defect; right hemianopsia. Perimeter charts.

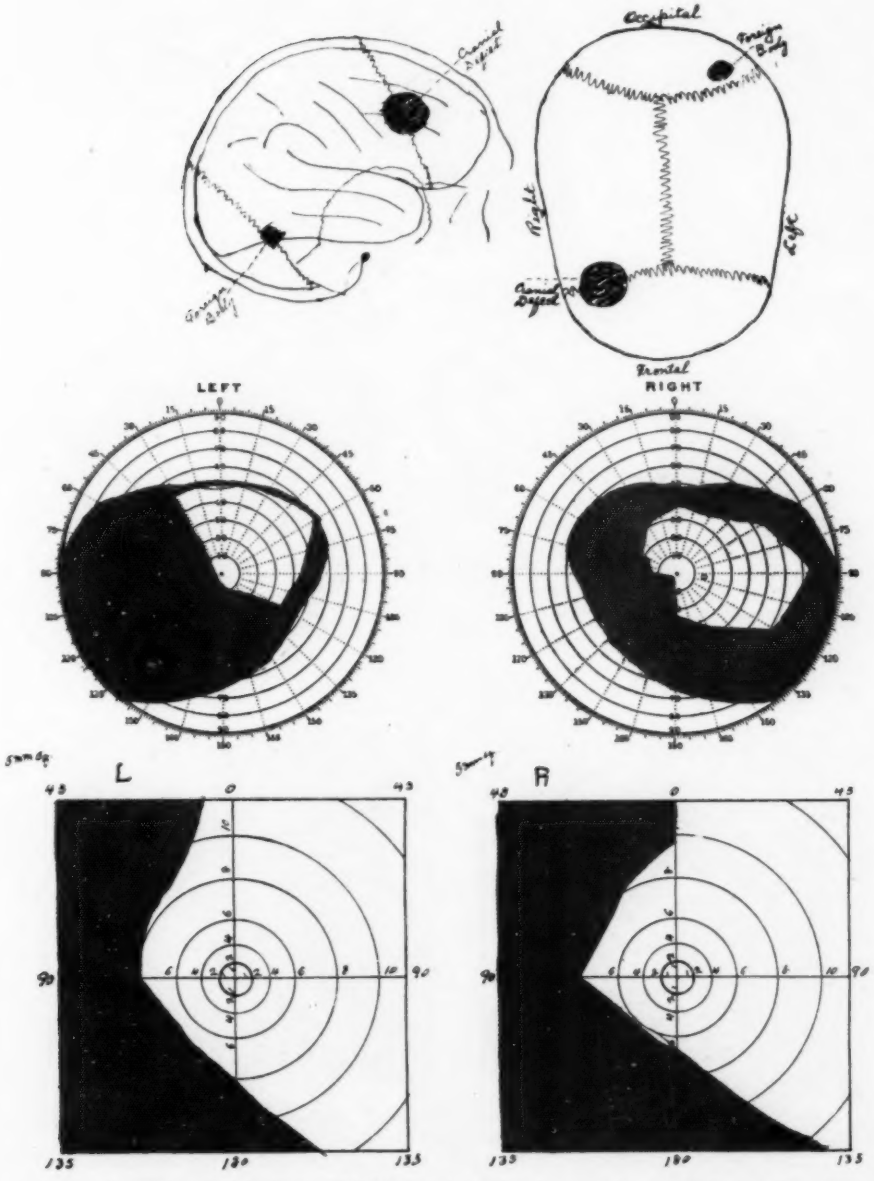

Fig. 7 (Case 7, M. D.).—Right frontoparietal wound and defect. Foreign body in left occipital lobe; right hemianopsia; dementia. Perimeter charts.

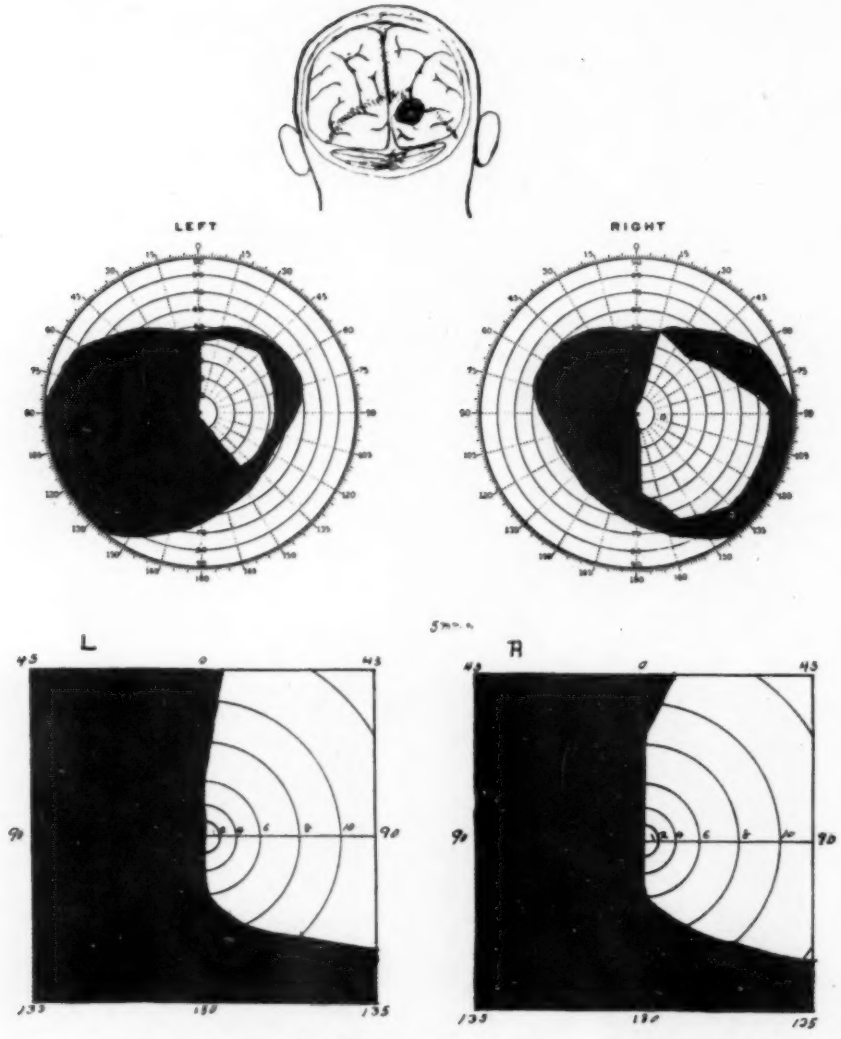

Fig. 8 (Charts 8, F. D.).—Right occipital wound and defect; left hemianopsia. Perimeter charts.

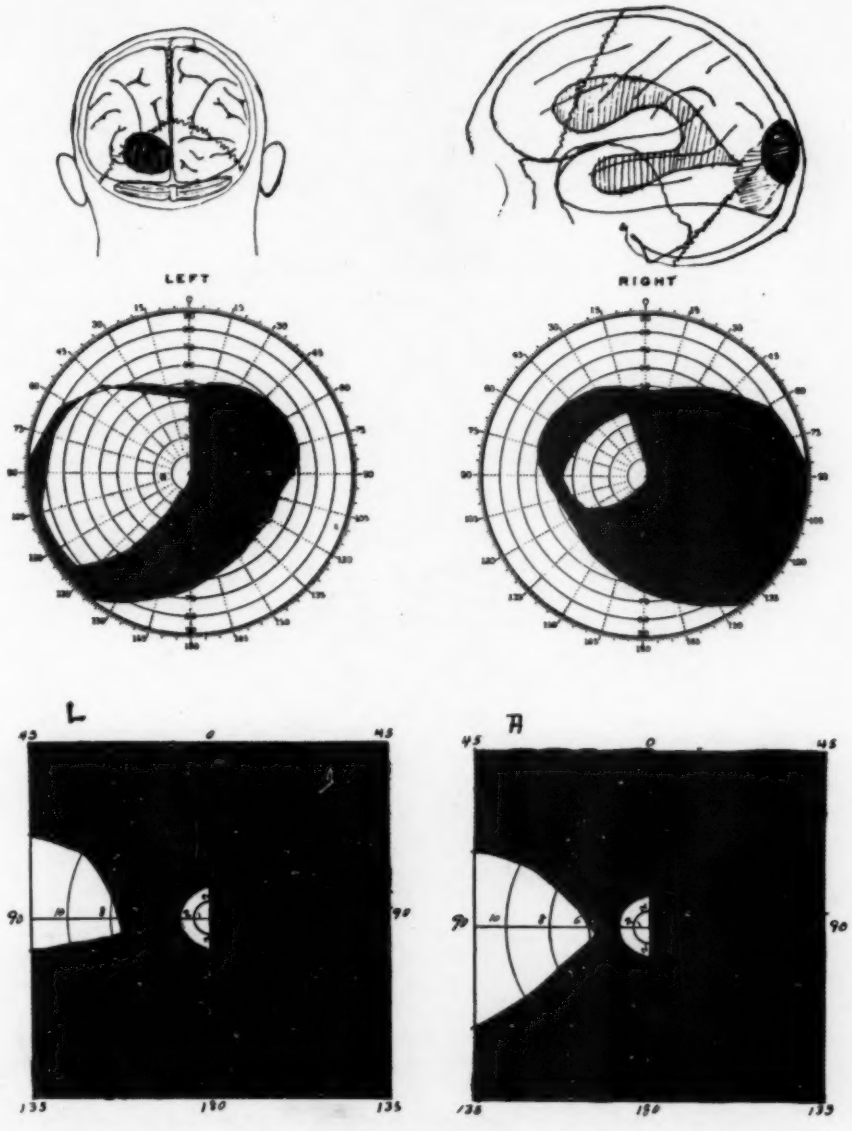

Fig. 9 (Case 9, L. S.).—Left occipital wound and defect; right hemianopsia and impairment of the left fields. Lateral aspect showing outlines of cyst cavity and air filled lateral ventricle. Perimeter charts.

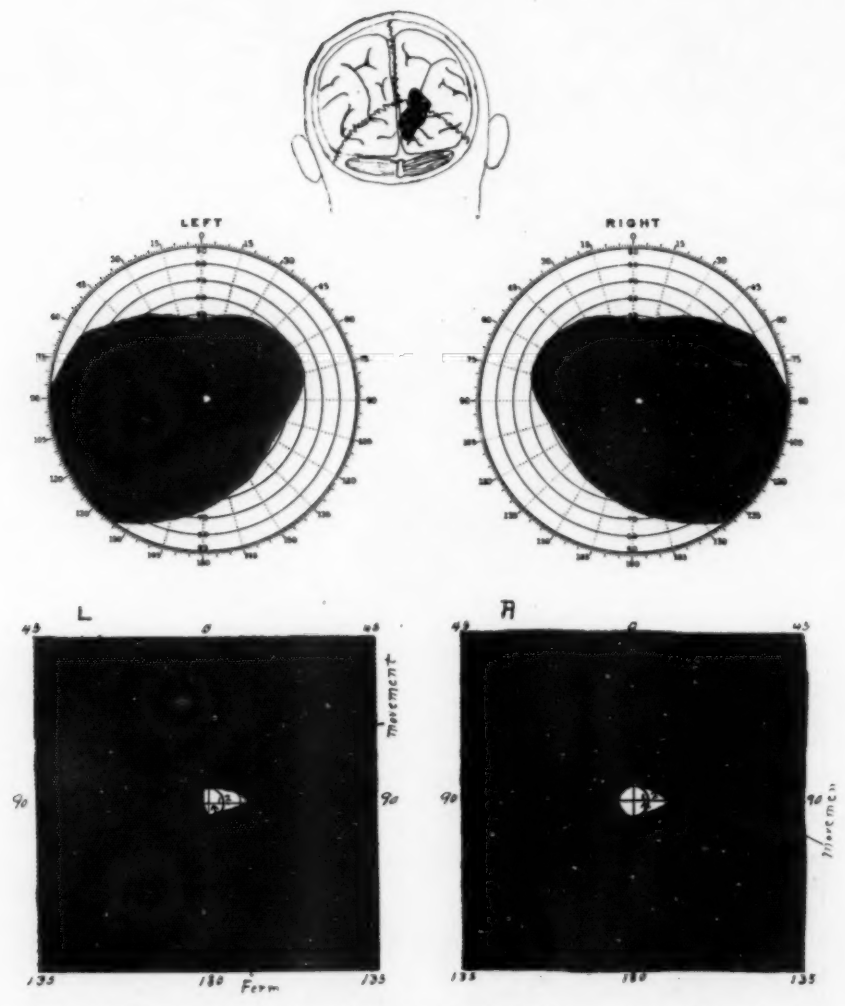

Fig. 10 (Case 10, B. H.).—Right occipitoparietal wound and defect. Complete left hemianopsia; incomplete right hemianopsia. Perimeter charts.

anopsia, and also defective left fields (Fig. 9). Macular vision was preserved in a semicircular area extending from a vertical line through the fixation point into the left fields 1.5 degrees. Roughly symmetrical islands of retained vision were present in the left fields but distinctly separated from the maculae. In order to study the hemianopsia in relation to the fixation points tests were made at a distance of 5 meters from the patient with a white object 5 mm. in diameter. At this distance and with steady fixation the object was seen only when it approached within 1 cm. on the right side of the fixation point on testing the right eye, and within less than 2 cm. when testing the left eye. These tests were repeated on several occasions, and the results were always definite and consistent.

Case 10.—B. H., was wounded, Oct. 4, 1918, in the right occipitoparietal region, after which he was unconscious for two weeks. He was admitted to Hospital No. 11, Dec. 22, 1918, the wound being unhealed. Roentgen-ray examination revealed a large cranial defect, 4 by 8 cm., involving the right side of

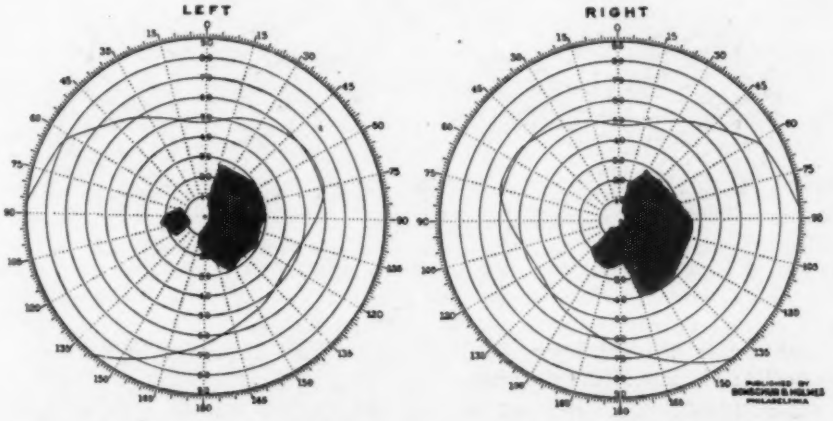

Fig. 11 (Case 11, V. M.).—Transverse tangential wound just below the inion. Irregular scotomas in right fields, but extending slightly into lower left fields.

the occipital lobe, and the right parietal bones (Fig. 10). The ocular movements, pupils, media and fundi were normal. Fixation was retained, and visual acuity was: right eye 20/40, left eye 20/40. Macular vision only was preserved, with the exception of the perception of bright light and large moving objects in part of the right fields. The retained macular areas were roughly cone shaped and symmetrical, extending from a semicircular line through the 1 degree point on the left of the fixation point of the right eye and the 0.25 degree point of the left eye toward the right for about 2 degrees.

Case 11.—On Oct. 14, 1918, the patient received a transverse tangential wound just below the inion. Immediately after the injury "everything looked white," but within one week the patient was able to distinguish objects. He was admitted to Hospital No. 11 in January, 1919. The pupils and fundi were normal, and there was a thin superficial opacity of the left cornea. Charts of the visual fields showed practically symmetrical scotomas of irregular outline, situated for the most part in the right fields, but extending to the left below the fixation point (Fig. 11). The macular areas were invaded to the fixation point, and central vision was reduced: right eye 20/70, left eye 20/100. The

left physiologic blind spot was abnormally large. The peripheral limits of both fields were approximately normal. The case is of interest, however, in correlating the scotomas with what was apparently a small cerebral lesion. From the fact that the acuity of central vision was reduced it is probable that neither occipital lobe remained entirely intact, the major portion of the scotomas occupying the right fields, and a minor portion the left fields, indicating a

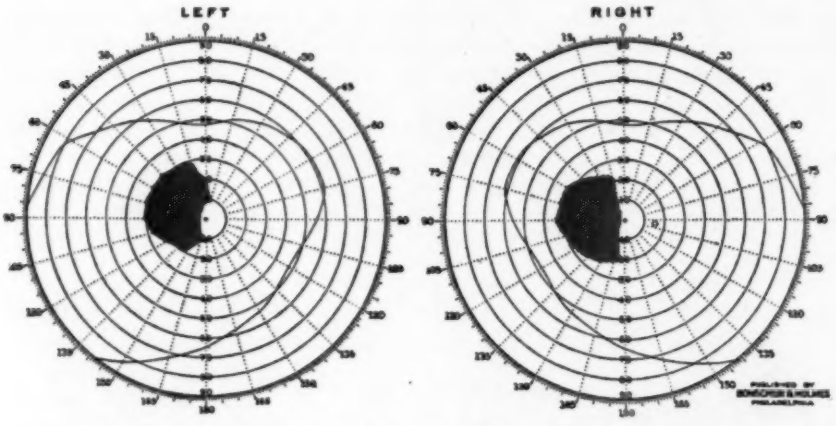

Fig. 12 (Case 12, F. E. G.).—Wound right occipital region. Symmetrical scotomas in left fields.

lesion of both occipital lobes, the left being affected to a greater extent than the right. Since complete hemianopsia was not present, neither occipital lobe was extensively involved. From the character and location of the wound it seems certain that any injury to the cerebrum must have been limited to the immediate vicinity of the occipital poles.

Case 12.—F. E. G. received wounds July 18, 1918, in the right occipital region. Early operation was performed in which bone fragments were removed.

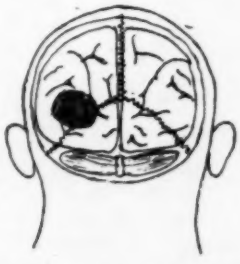

Fig. 13 (Case 13, T. C.).—Left parieto-occipital wound and defect. Homonymous lower right quadrant visual defect.

The patient was admitted to Hospital No. 11 on Nov. 6, 1918. On examination symmetrical scotomas were found in the left fields (Fig. 12) with normal fixation and visual acuity: right eye 20/20, left eye 20/20.

Case 13.—T. C. was wounded on Oct. 3, 1918, in the left occipitoparietal region. A roentgenogram showed a defect, 4 by 5 cm., involving the occipital and parietal bones (Fig. 13), and a linear fracture extending across the

parietal eminence from the defect. The charts of the fields of this case were lost, but it is recorded that both visual fields were defective in the lower right quadrants. The patient was right handed, and was able to read intelligently.

COMMENT

From the data obtained from the observation of this series of cases attention may be called to these points:

1. Fixation of vision and normal visual acuity (20/20 or better) are commonly retained when lesions of the occipital lobe have caused a complete homonymous hemianopsia (Cases 1, 3, 5, 6 and 8). In no case was there any indication that a unilateral occipital lesion caused a loss of fixation or of visual acuity, with the possible exception of Case 4 in which vision of the left eye was 20/30 (right eye enucleated). Error of refraction was not excluded as a cause of imperfect vision in this case. In all of the cases in which the character of the wounds indicated that both occipital lobes were injured, visual acuity was reduced in both eyes (Cases 7, 9, 10 and 11). No disturbance of fixation was demonstrated even in these cases.

2. The hemianopic fields commonly approached to within a fraction of 1 degree of the fixation point.

3. Evidence that macular vision is represented in the apex of the occipital lobe is furnished by Case 11.

4. Defects of the visual fields, scotomas or hemianopsias, resulting from lesions of the occipital lobes, are roughly symmetrical but not exactly superimposable.

5. Greatly reduced visual perception in homonymous fields (incomplete hemianopsia) may exist as a permanent residual result of occipital injury (Cases 2 and 10). In such cases bright lights and large moving objects can be discerned in fields which are blind to the usual tests for form and color.

In comparing the conclusions of Holmes and Lister with those of Morax, Moreau and others, it will be noticed that there are apparent contradictions in regard to the cortical representations of the macula. Holmes and Lister definitely state that the macula has not a bilateral representation, while Morax says that it is necessary to admit of a small zone at the point of fixation, probably 1 degree in extent, which possesses complex connections, since he has never found fixation affected in unilateral lesions. Visual acuity was not specifically mentioned by Holmes and Lister, but the observations of Morax and those of the authors show that central vision is commonly normal in cases of hemianopsia from occipital lesions, even when the blind areas extend to the fixation point. Morax makes a distinction between the "fixation point" and other parts of the macula, and uses the designations "area of precision" and "periphery of the macular area," from which it is clear that he conceives the macula as an area of high visual acuity, and

the fixation point as its center. If this conception be accepted, the charts of various observers indicate that half of the macular area is included in the hemianopic field; the median vertical boundary of the hemianopic field bisects the macula as well as the peripheral portions of the field. From this it may be concluded that each macular "area," as a whole, has bilateral connections in the cortex, the right half of each macula being represented in the right occipital lobe, and the left half in the left lobe.

Concerning the question of an overlapping of the retinal areas in relation to the right and left occipital lobes, respectively, certain evidence and analogies may be considered: 1. Fixation was never lost in unilateral occipital lesions. 2. In the cases most carefully charted it was constantly observed that vision was retained a fraction of a degree to the blind side of the fixation point. If the "fixation point" is a *fixed point*, a small overlap of innervation is indicated. 3. The well known overlapping of the sensory end-organs of the skin in adjoining peripheral nerve areas and spinal segment areas, and especially the sensory overlap along the entire midline of the body, each half of which is in relation with the opposite cerebral hemisphere, may be taken for analogies for an overlap of the retinal innervation. It appears plausible, therefore, that a very small overlapping innervation of the retina exists along the entire line of division between its lateral halves, and that the fixation point, situated on this line, actually possesses bilateral cortical representation as do all other points along the same line. This conception is apparently consistent with all of the phenomena observed in this series of cases, and with those of other observers.

CONCLUSIONS

1. Unilateral occipital lesions commonly result in homonymous hemianopsia, the blind field of each eye being limited by an approximately vertical line passing close to the fixation point.

2. Unilateral occipital lesions do not result in a loss of fixation nor a reduction of acuity of central vision of either eye.

3. Central vision is represented in the apexes of the occipital lobes.

4. Unilateral lesions at a distance from the occipital pole may result in approximately symmetrical paracentral scotomas.

5. Visual defects caused by lesions of the occipital lobes are approximately symmetrical but not exactly superimposable.

6. The macula is a central area of high visual acuity, not sharply circumscribed, extending a short distance in all directions from the fixation point which probably represents less than 1 degree in the arc of the visual field.

7. The hypothesis is suggested that a minute overlap of innervation exists along the entire vertical line separating the retinal halves. Each

half of the macula is thus in relation with the corresponding occipital cortex, and the fixation point, situated on the line of division, possesses bilateral cortical connections.

DISCUSSION

Dr. M. Allen Starr, New York: I should like to ask whether these fields of vision were symmetrical or whether there was very marked deviation in the limitation of the fields in the two eyes?

Dr. Charles W. Mills, Philadelphia: I think I was the first to point out that there may be a macular hemianopsia without a general peripheral hemianopsia. It seems unfortunate that in addition to the mere study of these fields, macular and peripheral, there was not also in the same cases a careful investigation of full and half vision for words, letters and special objects. This could readily have been done by a study of barrel or tubular vision.

There are probably lower and higher macular center fields in the cortex, the lower in some portion, probably in the posterior part of the calcarine region, and the higher in the lateral occipital lobe, probably in the angular region where Ferrier first placed them.

Dr. Edward A. Sharp, Buffalo: In using a plain dark screen, it is difficult for the patient to fix on it. Ten or fifteen feet back, the point of fixation is small. We use a long steel fishing rod end to carry the disk; but it is difficult at times to fix the object at the center. With the perimeter, we get closer to the object than with a screen. It seems to me that there is a chance for error in the fixation at a distance. Our difficulty has been in keeping the central point fixed at that distance.

Dr. M. Allen Starr, New York: The determination of the general visual field should be made differently from that of the field of vision of the macula, and the only satisfactory way of determining the latter is by having the patient look through a small tube which concentrates his actual fixation on a very bright point in the distance and then introduce the object at the end of the tube. In this manner, confusion, which comes from a large field, is eliminated.

Dr. Adolf Meyer, Baltimore: The perimeter examinations are probably the most difficult examinations that anybody can undertake with any patient. It seems to me that we must keep our minds and our eyes open to the relativity and to knowing to what extent we have the cooperation of the patient. In the studies that I have had an opportunity to make from the anatomic point of view, I have often deplored the fact that there could not have been some forethought by which points of anatomic interest might have been checked physiologically. The conclusion that I reached was that it was putting both the patient and the physician to the most difficult task to secure accurate perimeter examinations. The first thing is to get the closest estimate possible and then if there is any indication at all for more detailed examination, such measures as Dr. Starr mentioned, and other precautions, will have to exclude the distraction and the fatigability on the part of the patient and the physician.

Dr. Ingham, Los Angeles, in closing: In regard to the symmetry of scotomas and the defects in the visual fields, we found them to be quite consistent; that is, symmetry was present, not in the degree in which the one could be exactly superimposed on the other, but with a very close approximation. For instance, if the dividing line was at a slight angle, instead of being perfectly vertical,

the angle was similar in right and left fields. Gross cuts in the visual field were duplicated on both sides; but there was not a complete accuracy or perfect symmetry of the blind areas.

In reply to Dr. Starr's question, the size of the test object was, routinely, 5 mm., and the distance of the test 1 meter. A small white spot in the center of this screen served for fixation. The color fields, as well as the form fields, were charted and other methods were used, such as bright lights and large moving objects; but we found that for routine examination a white test object was most satisfactory.

Dr. Mills' reference to a hemianopsia of the macula is of interest, and we had one case in which this was demonstrated. There was a bilateral lesion, one visual cortex being completely destroyed and the other incompletely destroyed. Central vision was retained as well as a dim perception of large moving objects in a part of one field. Accurately charting the small central fields of vision revealed the characteristics of a bilateral hemianopsia of the macula. In other words, this patient had only macular vision in which was a hemianopsia.

Regarding the question of the higher and lower centers, the integrative functions in respect to vision came up for consideration. There was "word blindness" and "word deafness" in one of the cases and aphasic symptoms in others. These problems are not presented in this paper. We did not find any cases similar to those to which Holmes and Riddock referred, in which there was a loss of the stereoscopic vision without loss of visual perception.

The difficulties of fixation are quite a matter for consideration in the examination of patients. The perfectly black screen without any relief from the monotony does not enable some patients to maintain fixation. There are many cases in which fixation is difficult, especially when there is a degree of amaurosis of the central field. There was but little trouble in that connection in the cases here reported.

With reference to the quadrant defects, we had some examples of the quadrant blindness which were roughly symmetrical; and there was one case in which there was only little more than a quadrant of vision remaining in each field, a complete hemianopsia on one side, and a quadrant on the other side. Then there were scotomas, one paracentral scotoma, and another scotoma involving the macular area. These scotomas were also symmetrical in their situation and form but not exactly superimposable.

The difficulty of charting defects of the visual fields is very similar to the difficulty of charting areas of loss of sensation on the skin; the fatigue phenomena, the psychology of the patient and the psychology of the examiner must all be considered. All these points enter into the problem and should be checked carefully in making the record.

ELECTROMYOGRAPHIC STUDIES OF PARALYSIS AGITANS *

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For many years graphic records of various tremors have been made by means of tambours and levers. Only recently has the string galvanometer been employed to make such records, with obvious advantages. Not only is greater accuracy possible, but the recording of the action-currents gives an indication of the physiologic activity within the muscle during the periods when the muscle is mechanically quiescent. This accuracy, moreover, emphasizes the remarkable constancy of the periodic phenomena of tremor, and draws our attention to the principles which must underlie these neuromuscular rhythms.

Electromyography, or the study of the action-currents of the skeletal muscles, was first described from the physiologic standpoint by Piper,[1] the interpretations being later modified by Forbes and Rappleye.[2] A short description of the method as applied to the study of paralysis agitans may be inserted here.

In order to record the electrical changes in a contracting muscle a continuous circuit must be established through that muscle and the string of the galvanometer; thus there must be two electrodes on the body surface, one connected with each end of the string. One of these is applied to the skin over the belly of the muscle to be studied, and the other is placed over some nearby skin area beneath which there is no muscular contraction. In studying the tremor of paralysis agitans, the muscle usually recorded was the flexor carpi radialis because of its frequent involvement in the tremor and its convenient location for the application of electrodes. One of these is placed directly over the belly of this muscle, the other (the "indifferent" electrode) may be placed on any other part of the body, but in order to avoid action-currents from the heart it is best to have it on the same limb; and in order to avoid action-currents from other contracting muscles it should be placed over tendons or over some completely inactive muscle. The flexor surface of the wrist is a good place, but if the tremor is

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* From the medical laboratories and neurological service of the Massachusetts General Hospital.

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1. Piper, H.: "Elektrophysiologie menschlicher Muskeln," Berlin, 1912.
2. Forbes and Rappleye: Am. J. Physiol. **42**:228, 1917.

marked there will be movement of the electrode and mechanical shifting of its position on the skin, causing long waves in the record (Plate 4, Fig. 1). It is therefore often better to rest the elbow and weight of the arm on a table; with the biceps and triceps thus relaxed the "indifferent" electrode may be placed on the outer surface of the lower arm. This completes the circuit through body and galvanometer, and the record as nearly as possible represents the action-currents set up by the contractions of one muscle—in this case the flexor carpi radialis. It must be remembered, however, that in the tremor of paralysis agitans many muscles are involved and may be contracting at different times; even from a distance these may give lesser action-currents to complicate the picture.

The apparatus employed in these studies was the string galvanometer designed by Dr. H. B. Williams and manufactured by C. F. Hindle & Co. For electromyographic studies a galvanometer and recording camera such as are used for cardiographic work are perfectly satisfactory. Most of our records were obtained with the regular electrocardiographic equipment of the hospital. We were able to use this apparatus by simply increasing the speed of the falling plate and by supplying our own special electrodes. The camera generally used was of the type manufactured by the Cambridge Scientific Instrument Company, the speed of the plate being increased from the 3 cm. per second used to take electrocardiograms to 5 or even 10 cm. per second; the most satisfactory speed for this work being between 5 and 6 cm. per second. A few of the cases were studied at the Harvard Medical School in the laboratory of Dr. Alexander Forbes, where there is a camera of special design[3] which uses moving picture film and gives high rates of speed with remarkable uniformity.

At the hospital I used small German silver electrodes, one with a surface area of 100 sq. cm., the other of 50 sq. cm. Beneath these were placed pads moistened in hot physiologic sodium chlorid; electrode and pad were then firmly bandaged to the limb. In Dr. Forbes' laboratory we used nonpolarizable zinc-sulphate electrodes.[4] These are more difficult to apply to the patient, but they keep a more uniform moisture and resistance and seem to give better definition to the small action-currents, especially when a high-speed film is employed for recording.

With the patient in position the electrodes are firmly fastened to the appropriate spots. The galvanometer is then set with the string at such a tension that 1 millivolt causes a deflection of 1 cm. on the

3. Forbes and Thacher: Am. J. Physiol. **52**:409 (July) 1920.

4. Cobb, S.: Bull. Johns Hopkins Hosp. **29**:247 (Nov.) 1918. Also foot notes 1 and 2.

record. The connections are made with the patient; the resistance of the patient is measured and compensated, and the camera and lights are arranged for the making of the record.

OBSERVATIONS

By this method eighteen cases of tremor were studied. All of these showed features of the paralysis agitans syndrome, and in all the tremor was of the coarse type found in that condition, but only eleven of the cases were typical Parkinson's disease or the senile form of paralysis agitans. Case 17 is probably a case of early Parkinson's disease, not yet definite enough to be diagnosed; the patients in Cases 2, 5 and 9 almost certainly had basal ganglion lesions and they had tremors of the paralysis agitans type, but their clinical pictures were confusing, Case 5 being more of an athetoid chorea with tremor. Cases 10 and 12 were definitely examples of the paralysis agitans syndrome following lethargic encephalitis, while Case 14 may have been postencephalitic.

REPORT OF CASES

CASE 1.—C. A., a woman, aged 59, was seen Oct. 15, 1919. Three years before her right ankle was injured. In May of this year (five months before) she first noticed shaking of her right arm and of the right side of her face, with a peculiar weak feeling in her knees. Examination revealed the fact that the right arm was so stiff and weak that she could hardly raise it to her mouth. Walking was difficult only in getting started; when she was once "under way" there was no further trouble. There was constant tremor of the head and of the right arm. Occasionally this tremor was noticed in the left hand. Any excitement increased this tremor, which was of the parkinsonian type. There was rigidity of the muscles of the right arm. The knee reflexes were hyperactive. Mental examination showed marked depression.

The patient died in July, 1920.

Diagnosis: Paralysis agitans.

CASE 2.—J. B., a woman, aged 33, seen in May, 1919, for six weeks had had aches in her left wrist. There was tenderness over the styloid process of the radius and she said that a year ago she had had acute swelling in this location as well as in the left leg. For six months there had been increasing tremor of the left hand. The knee reflexes were exaggerated. There was some atrophy of the muscles of the left hand and the tremor, which seemed to be in this hand only, was exaggerated by putting the muscles on slight tension; for example, pressing a little against the fingers of the extended hand brought it out conspicuously. In this respect it resembled a clonus, but the spontaneous tremor seen when she tried to hold a glass of water was of practically the same rate. There was slight weakness of the left side of the face and marked enlargement of the thyroid. In September, 1921, the tremor was present in both hands and both legs. There was some rigidity of the muscles and pain in the left knee. The blood Wassermann reaction was negative.

Diagnosis: Tremor resembling that of paralysis agitans in a patient with arthritis, who later developed a more typical paralysis agitans picture.

Case 3.—A. B., a man, aged 60, was seen July 11, 1921. Four years before the left thumb showed slight tremor which gradually increased and progressed until the hand and arm were involved. Any motion increased this tremor. After two years the same kind of tremor began to develop in the right leg, and one year ago stiffness of the muscles of the right arm was noticed, while rigidity of the legs appeared six months ago. At the time of the first examination, the patient showed the typical parkinsonian attitude and gait with "pill rolling" tremor, more marked on the right. The face was masklike and the eyes staring. There was slight propulsion. In October, 1921, examination revealed in addition weakness of the left arm with a slightly flexed position of the fingers and weakness of the left leg. The patient has been treated (Table 2) for about seven months and at present (April, 1922) shows less rigidity and considers himself more comfortable, but otherwise he shows no evidence of change. The Wassermann reaction was negative.

Diagnosis: Paralysis agitans.

Case 4.—J. S. B., a negro, aged 48, was seen Dec. 30, 1919. The first symptom was stiffness of the right arm and hand in May, 1919. In September tremor of the right hand and both sides of the face developed. There were periods when the whole body seemed stiff, particularly noticeable in the legs. On admission the patient showed marked speech defect, a masklike face with drooping of the right side; the right hand had a conspicuous coarse tremor which was increased when he was excited but practically stopped when he was distracted. He could control this tremor sufficiently to write or draw straight lines on a blackboard. In April, 1920, he showed more speech defect, otherwise the condition was the same. In March, 1922, there was no change except that the right side of the face was more conspicuously drooping and the tremor was, if anything, greater. The blood Wassermann reaction and lumbar puncture were negative.

Diagnosis: Paralysis agitans.

Case 5.—A. C., an Italian girl, aged 12, was seen March 15, 1917. For some years she had had a skin ulceration diagnosed as tuberculous. In the middle of the winter of 1917 she developed chorea. At this time she had a spasmodic jerking motion of the left hand and arm that was somewhat rhythmic, the rhythm interrupted by an occasional slashing motion of the left arm of a choreic type. The left leg was spastic, and she walked with a hemiplegic gait. One year later she showed considerable improvement in the tremor in the right arm, but it was still easily elicitable by asking her to put the arm on slight tension. There was also fine tremor of the tongue and occasionally athetoid movements of the left arm and leg. Speech was staccato. The tendon reflexes were slightly exaggerated. There was a Babinski sign on the left. The Wassermann reaction was negative.

Diagnosis: Athetoid-chorea with parkinsonian tremor.

Case 6.—C. C., a woman, aged 51, was seen Nov. 14, 1919. In 1916, she noticed difficulty in using the right hand and a tendency to carry the right arm partly flexed. There was also a dull ache in the right shoulder and breast. About a year and a half after the onset of these symptoms the right arm and leg began to tremble. Examination revealed continuous coarse tremor of the right arm and leg; anything which caused increased muscular tension increased the tremor. On account of the pain and stiffness in the right shoulder she

was unable to raise her right arm high enough to comb her hair. "It is exactly half of my body which is affected." The blood Wassermann reaction was negative.

Diagnosis: Paralysis agitans.

CASE 7.—K. C., a woman, aged 58, was seen May 29, 1919. For six months this patient had had tremor of the right hand and arm. She could use her hand very well, and there was no stiffness. The knee reflexes were present. In July, 1919, there was little change, but in January, 1920, a year later, the facial expression was somewhat masklike and the right side of the face looked flatter. The tremor consisted of pronation and supination of the right hand, with little if any flexion and extension of the fingers. The right leg and the whole left side were unaffected. In walking the right hand showed tremor, the arm was slightly flexed at the elbow and swung slightly but distinctly less than the left one. In January, 1920, the tremor was more marked, but there was no masklike expression. The face burned, but there were no other sensory symptoms except that the left hand sometimes felt as if "asleep." The blood Wassermann reaction was negative. The blood calcium was 8.5 mg. per 100 c.c. plasma.

Diagnosis: Paralysis agitans.

CASE 8.—M. D., a woman, aged 60, was seen Oct. 7, 1919. She had noticed coarse tremor of the right arm suddenly in April, 1917. She had had eighteen children. She had always been nervous, but since having the influenza the preceding winter she had been more unstable than ever. Examination revealed widely dilated pupils and exaggerated tendon reflexes. There was slight tremor of the right arm, most marked in the flexor and extensor muscles of the wrist and fingers. The blood Wassermann reaction was negative.

Diagnosis: Paralysis agitans.

CASE 9.—M. A. D., a girl, aged 14, was seen March 13, 1915. One year before she first complained of shaking of the left hand and inability to grasp objects properly. Examination on admission revealed ptosis of the left lid, slight enlargement of the thyroid, wide pupils, exaggerated tendon reflexes and marked tremor of the left arm and hand. Tonsillectomy was performed, and she was discharged. She returned in October, 1918. She reported that the tremor in the left arm had gradually become worse, with numbness in both the left arm and leg. Occasionally there was pain in the left side. The left arm and leg were somewhat weak and her gait slightly hemiplegic. In October, 1919, the condition was similar but somewhat worse, with more pain and more tremor. The blood Wassermann reaction was negative. The basal metabolism was two plus.

Diagnosis: Parkinsonian tremor with atypical syndrome.

CASE 10.—E. E., a man, aged 18, was seen March 4, 1922. Two years before, after recovering from pneumonia, he noticed slight difficulty in speech. This became more marked following an accident in which a can of red lead was said to have fallen and struck him on the back of the neck. A few days later he noted twitching movements in the arms and legs which gradually increased until, four months later, he gave up work on account of lack of control of his hands. This passed off in about four months. Thirteen months after the injury (seven months ago) he began to feel pain in the back of the neck radiating down to the shoulder blades. Six months ago his legs began to be rigid and his gait unsteady. With the development of these symptoms tremor

in the left arm was noticed, and the facial expression became masklike. More recently his fingers have become numb. At present he is bedridden and lethargic, but when aroused shows good memory and intelligence. The Wassermann reaction was negative on the blood and spinal fluid. The spinal fluid pressure was 285 mm. of water; otherwise negative. Four determinations of the basal metabolism were: plus 15.5, plus 21, plus 5 and plus 27.

Diagnosis: Lethargic encephalitis.

Case 11.—M. P. G., a man, aged 56, was seen in June, 1919. Four years before he noticed slight trembling in both hands, but more on the right. He was unable to write as well as formerly, and it was hard to bring a fork to his mouth. Physical examination showed a masklike face, loss of associated movements of the arms and legs in walking, the typical parkinsonian attitude in standing and coarse tremor of both hands. This tremor was somewhat more marked on the right. In both legs a slight tremor was palpable, and he complained of muscular cramps. The blood Wassermann reaction was negative.

Diagnosis: Paralysis agitans.

Case 12.—T. I., a boy, aged 8, was seen April 12, 1922. The past history was negative. In February, 1920, the patient had a croupy cold, after which he became sleepless, talkative and hyperactive, with twitchings of the arms, legs and head. He complained of diplopia. Two or three months after recovering from the acute symptoms, he had two convulsions and began to show rigidity and tremor, more marked on the right. The facial expression became masklike, the speech monotonous and tremulous. After this he improved; the tremor and twitchings became less marked. Three months ago he became worse; he lost the use of the right arm and leg to some extent and the tremor on the right side grew worse. He became very unruly. His head was usually held to the right, the right leg was spastic, and he walked on the toes of the right foot, planting it heavily. The deep reflexes were decreased on the right, normal on the left. The picture was one of masklike facies, propulsion, parkinsonian attitude, tremor, rigidity of the arms and rigidity of the legs without tremor, more marked on the right side.

Diagnosis: Paralysis agitans syndrome following lethargic encephalitis.

Case 13.—M. K., a man, aged 39, was seen in July, 1907. The patient was referred to us because of tremor of the right hand. In 1919 he reported back and said that during the last twelve years this shaking had been increasing gradually until the right hand could be stopped only by grasping it with the left. Occasionally there was also a tremor in the left arm and a quivering of the face around the mouth, and the protruded tongue. The face was masklike and speech slow. Tendon reflexes were exaggerated. The blood Wassermann reaction was negative.

Diagnosis: Paralysis agitans.

Case 14.—Mary K., a woman, aged 43, was seen Feb. 11, 1920. For a year and a half she had noticed slight trembling in the left leg and for four months in the left arm. During the last few days the head had begun to feel tremulous. Physical examination revealed slight edema of the left leg and of the left arm, with an area on the skin over the triceps that felt as if the skin were thickened and infiltrated. There was no rigidity of the muscles, and the facial expression was normal. The left hand had a slight tremor of parkinsonian type. The left leg also, when balanced on the ball of the foot, showed tremor. The blood Wassermann reaction was negative.

Diagnosis: There is a possibility that this case represents a residuum from lethargic encephalitis because an indefinite history of a period of coryza and somnolence was obtained, but it appears more probable that it was an incipient case of paralysis agitans.

Case 15.—G. H. M., a man, aged 56, was seen Jan. 6, 1920. Three years before tremor started in the left hand and one year later in the left leg. This was accompanied by a dull ache in the neck. Examination revealed coarse tremor of the left arm and left leg when the muscles were put on slight tension. The muscles of the trunk were also slightly involved on the left; for example, there was a tremor in the pectoralis major and the latissimus dorsi. The tendon reflexes were somewhat exaggerated. The muscles of the left arm showed slight rigidity, with a "cogwheel" feeling on passive motion. The left side of the face appeared somewhat flattened and expressionless. The blood Wassermann reaction was negative.

Diagnosis: Paralysis agitans.

Case 16.—R. M., a woman, aged 42, was seen Dec. 20, 1918. Two years before she noticed shaking in the left arm, but the onset was insidious. A few months afterward, the left leg was affected in the same way when she put her weight on it. The left leg and arm were weaker than the right, and the tendon reflexes on this side seemed to be somewhat more lively. There was marked tremor of the left hand, and arm and of the protruded tongue. The right hand showed a fine tremor. The skin of the left hand was distinctly smooth and shiny. The left leg showed no skin changes, but a marked tremor when the weight of this extremity was balanced on the ball of the foot. The left side of the face was slightly flattened and less expressive than the right. The blood Wassermann reaction was negative. The spinal fluid showed 8 cells, but was otherwise negative.

Diagnosis: Paralysis agitans.

Case 17.—M. V. S., a woman, aged 43, was seen July 26, 1919. In May of this year she noticed a prickling sensation in her left forefinger. This later spread to the thumb and the third finger. She showed slight tremor of the right hand when extended and of the left hand when at rest, but this was stopped by voluntary motion. There had been some numbness of both legs, more marked on the right. All four extremities were stiff. The blood Wassermann reaction was negative.

Diagnosis: Tremor of the parkinsonian type in a neurotic person.

Case 18.—J. T., a man, aged 57, was seen April 15, 1920. Six years before he had been jammed against the side of a door by a horse and had received a minor injury of the right shoulder with difficulty in walking. Six months after this there was marked trembling of both legs when he attempted to walk. Four years ago the right hand started trembling and a few months after that the left hand. The face showed the typical masklike appearance, and the voice was monotonous. There was coarse tremor of both hands with the "pill rolling" motion, numbness in the legs and arms—more marked on the right—and loss of association of the movements of the arms and legs in locomotion. The blood Wassermann reaction was negative. The basal metabolism, in two determinations was plus 23 per cent. and minus 2 per cent.

Diagnosis: Paralysis agitans.

In Table 1 the cases are arranged in alphabetic order and the data of each category are entered in the appropriate column. It will be

TABLE 1.—Findings in Author's Cases

Case Number	Initials of Patient	Date of Observations	Rate of Tremor per Second	Frequency of Action-Currents of Tremor-Contractions per Second	Frequency of Action-Currents Between Tremor-Contractions per Second	Muscle from Which Action-Currents Were Recorded	Approximate Length of Muscle in Centimeters	Heart Rate	Age	Sex
1	C. A.	10/22/19	5.6	45	65	Extensor pollicis brevis	17	..	59	F
2	J. B.	8/ 4/19	5.1	60	80(?)	Extensor carpi ulnaris	22	..	33	F
		10/ 2/19	5.4	55	75	Extensor carpi ulnaris	22	..	33	F
3	A. B.	3/29/22	5.2	40	65	Flexor carpi radialis	27	82	61	M
4	J. S. B.	12/30/19	4.9	50	75	Extensor communis digitorum	42	70	48	M
		12/30/19	4.9	..	90	Flexor carpi radialis	31	70	48	M
5	A. C.	7/ 7/19	4.8	..	90	Flexor carpi radialis	26	..	14	F
		7/14/19	5.0	55	80	Flexor carpi radialis	26	..	14	F
6	C. C.	11/14/19	6.4	40	70	Gastrocnemius	35	..	51	F
7	K. C.	1/29/20	5.8	50	75	Flexor carpi radialis	28	..	58	F
8	M. D.	9/30/19	5.4	50	75	Extensor communis digitorum	37	..	60	F
		10/ 7/19	5.1	..	..	Extensor communis digitorum	37	..	60	F
9	M. A. D.	10/16/19	4.7	..	60	Flexor carpi radialis	28	90	18	F
		10/20/19	5.0	50	75	Extensor communis digitorum	38	..	18	F
10	E. E.	3/ 4/22	5.6	..	..	Triceps	36	78	18	M
		3/20/22	5.5	25	53	Triceps	36	90	18	M
		3/20/22	5.4	40	55	Triceps	36	90	18	M
11	M. G.	6/20/19	6.5	55	80(?)	Abductor pollicis brevis	7	..	56	M
12	T. I.	4/12/22	9.0	50	105(?)	Extensor communis digitorum	26	130	8	M
13	M. K.	1/29/20	6.4	30	60	Flexor carpi radialis	30	..	52	M
		11/16/21	6.5	40	65	Flexor carpi radialis	30	100	53	M
		12/ 5/21	6.0	..	..	Flexor carpi radialis	30	98	53	M
14	M. K.	2/11/20	6.6	..	..	Gastrocnemius	35	..	43	F
		2/11/20	6.6	..	..	Gastrocnemius	35	..	43	F
15	G. H. M.	1/ 6/20	6.1	45	70	Flexor carpi radialis	29	..	56	M
		1/ 6/20	5.7	..	..	Gastrocnemius	36	..	56	M
16	R. M.	10/ 2/19	5.8	60	90	Gastrocnemius	35	73	43	F
		11/ 4/19	6.2	60	95	Gastrocnemius	35	73	43	F
17	M. V. S.	7/26/19	6.1	40	85	Flexor carpi radialis	27	..	43	F
18	J. T.	7/31/20	6.0	..	..	Flexor carpi radialis	29	..	57	M
Average..	.....	.....	5.8	46	73					

seen that two kinds of measurement were made from each record and their different natures must be clearly distinguished: first, the rate of tremore indicates the number of short, clonic contractions per second made by the muscle involved. A tremor of this kind consists of a series of muscular contractions following each other at a rapid and more or less constant rate; each of these contractions we call a tremor-contraction. Now, all biologic activity is accompanied by changes in electrical potential which set up action-currents; it is these action-currents which are recorded by the string galvanometer, and when led off from a muscle they make an electromyogram. If such a record is compared with a mechanical myogram it is found that the action-currents of any given contraction occur just previous to and with the beginning of the mechanical contraction of the muscle.[5] Therefore the electromyogram may be used to record the time relations of muscular contractions. In examining these electromyograms of the tremor of paralysis agitans it is seen that with each tremor-contraction there are one or more large diphasic action-currents; the usual number is from two to four. After this group of action-currents the string is less active, but in this disease is rarely at rest. In other words, between the tremor-contractions there is some muscular activity in progress which is represented in the electromyogram by a series of small, rapid waves, whereas the waves of the tremor-contraction group are larger and slower. Thus in Table 1 there are two columns for action-current frequencies, one the frequency of action-currents of the tremor-contraction per second, and the other the frequency of action-currents between the tremor-contractions per second. It is possible that these different forms of action-currents represent different physiologic processes, and the fact that the frequency of action-currents between the tremor-contractions is constantly higher than that of the action-currents of the tremor-contractions is in favor of this view, the average for the former (the sixth column of Table 1) being 73 per second and for the action-currents of the tremor-contraction (fifth column) being 46. Such a distinction immediately brings to mind the double symptomatology of paralysis agitans—tremor and rigidity—and one is tempted to say that the electromyogram depicts these two phenomena by these two types of action-currents. But before stating such a theory much more investigation should be made of the nature of rigidity. Electromyograms of decerebrate rigidity[6] in cats show fine rapid action-currents at a frequency of 70 to 90 per second comparable to those seen in our records between the tremor-contractions. This analogy may be significant, but

5. Salomonson, J. K. A. W.: Brain **43**:369, 1920.

6. Buytendyk, F. J. J.: Ztschr. f. Biol. **59**:36, 1912. Einthoven, W.: Arch. Neerland. de Physiol. **2**:489, 1918.

there are so many unsettled questions concerning rigidity, spasticity and tonic contraction that I do not wish to go into the discussion of tonus and tonic innervation. There is much conflicting information on the subject and new evidence must be collected before these most important phenomena are understood. For example, Salomonson [5] says that muscle contractions may be classified in three different groups: (*a*) the simple twitch which gives a single diphasic action-current; (*b*) the voluntary contraction with its tetanic alternating action-currents at the rate of about 50 per second, and (*c*) the tonic muscular contractions in which "we may observe strong muscle spasms and also notable changes of the muscle tension without any electric action-current."

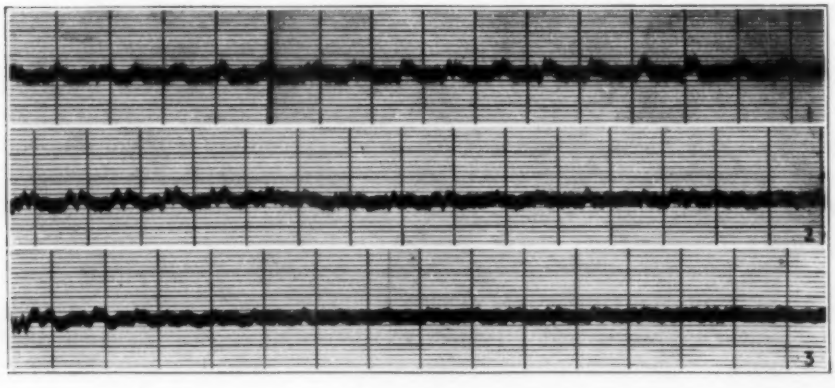

PLATE 1

Paralysis agitans syndrome following encephalitis, Case 10. Time marker, 1/5 of a second. The patient showed a slight inconstant tremor and marked rigidity.

Fig. 1.—Electromyogram from triceps of right arm showing tremor at rate of 5.4.

Fig. 2.—Triceps as in Figure 1. At the beginning there is tremor which ceases, leaving rigidity. Action-currents at frequency of 53 per second.

Fig. 3.—Electromyogram of triceps at rest with no tremor, but marked rigidity. Main waves at frequency of about 45 per second.

The first two groups are well substantiated by physiologic and clinical studies with the string galvanometer since the original researches of Piper.[1] But the nature of tonic muscular contraction cannot be disposed of so summarily. As noted in the foregoing, Salomonson considers that these tonic contractions are accompanied by no action-currents, whereas Hoffmann [7] believes that tonus is a weak tetanus. He is corroborated by Rehn,[8] who made electromyograms of spastic

7. Hoffmann, P.: Arch. f. Physiol., 1913, p. 23.
8. Rehn, E.: Deutsch. Ztschr. f. Chir. **162**:155. 1921.

muscles before and after operations. This raises the question as to whether the decerebrate rigidity of experimental animals [9] is comparable to the extensor rigidity seen clinically in man. Walshe [10] considers these practically identical, and we have the evidence quoted that both give rapid tetanic action-currents, although Salomonson holds that spastic and rigid phenomena in the human are tonic phenomena and produce no action-currents, stating [11] that he finds great muscle spasm in diseases of the corpus striatum and regio subthalamicus which do not show any action-currents.

Case 10 of our series may be taken as evidence on this point. The patient showed great rigidity of the arms and occasional periods of coarse tremor, especially marked at the elbow. The electromyograms were made from the triceps (Plate 1). Figure 1 shows a typical paralysis agitans tremor at the rate of 5.4 per second. Figure 2 shows the tremor for one second, and then a period of two seconds in which the tremor fades out and disappears, leaving a tetanic series of action-currents at a frequency of 53 per second. In Figure 3 we see no evidence of the regular tremor, merely a series of small waves at a frequency of 45 or 50 per second. All of these records were made from the triceps, the electrodes being unchanged during the series. The arm was at rest, the patient being supine with shoulder, elbow and wrist supported on pillows. Electromyograms of normal muscles thus put at rest show no electrical activity when studied with this apparatus.

Although these observations may seem to indicate that the rigidity of paralysis agitans is accompanied by a tetanic series of action-currents, we must remember that certain artefacts possibly account for the electrical variation. First, distant muscles other than the triceps might be contracting and transmitting action-currents to the electrodes. It is impossible to lead off the action-currents from one part of the body alone. Second, oscillations of large amplitude would render slightly more difficult the detection of small superimposed waves. Moreover, many of the records were not made rapidly enough and with accurate enough focus to give definition to their fine waves. But enough have been measured and placed in the sixth column of the tables to indicate that on the average this rate is 59 per cent. more rapid than the action-currents of the tremor-contractions.

The best example of this contrast between tremor-contraction and rigidity is seen in Case 13 (Plate 2, Fig. 2). Here the large tremor-contraction waves are definite and slow at a frequency of 38 per second, whereas the small waves between are equally distinct, but at

9. Sherrington, C. S.: Brain **38**:191 (Nov.) 1915.
10. Walshe, F. M. R.: Brain **42**:1 (April) 1919.
11. Salomonson, J. K. A. W.: Brain **42**:372, 1919.

a frequency of 70 per second. Plate 2, Figures 1 (Case 3) and 3 (Case 12) show the same thing registered on a more rapidly moving film.

The tremor of paralysis agitans, as recorded by this method, shows some interesting points. In the first place, the tremor appears to be due to periodic, short muscular contractions, each one of which is accompanied by a series of from two to six or eight relatively large action-currents (Plate 2, Figs. 1, 2, 3, 4, 5). That is to say, each one may be looked on as a short tetanic contraction unlike the contraction

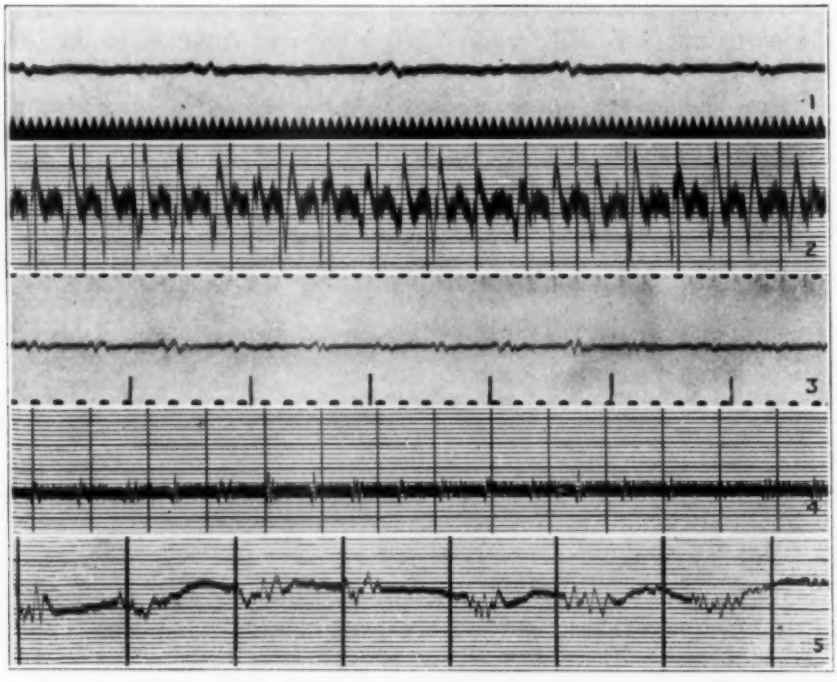

PLATE 2

Types of electromyograms in paralysis agitans.

Fig. 1 (Case 3).—(Dr. Forbes' apparatus, greater speed and tuning fork at 1/100 of a second). Between large waves (at frequency of 47 per second) which precede each tremor-contraction are smaller waves (at frequency of 86 per second); tremor rate 5.1.

Fig. 2 (Case 13).—For comparison with Figure 1. Frequency of main waves of tremor-contractions about 38 per second; frequency of main waves between tremor-contractions 70 per second; rate of tremor 6.2.

Fig. 3 (Case 12).—(With Dr. Forbes' apparatus.) Showing rapid rate of tremor in a child; rate, 9 per second. Twin marker, 1/5 second.

Fig. 4 (Case 16).—Typical myogram for paralysis agitans.

Fig. 5 (Case 5).—A typical curve of mixed chorea-tremor case.

TABLE 2.—Data from Case 3—Patient A. B.—Over a Period of Five Months Under Varying Conditions and from Five Different Muscles

Date of Observations	Rate of Tremor per Second	Frequency of Action-Currents of Tremor-Contractions per Second	Frequency of Action-Currents Between Tremor-Contractions per Second	Muscle from Which Action-Currents Were Recorded	Approximate Length of Muscle in Cm.	Heart Rate	Age of Patient	Special Conditions
10/28/21	5.3	50	75	Extensor pollicis brevis	18	80	60	Sodium cacodylate, 12 grains four times a day
10/28/21	5.2	35	60	Extensor pollicis brevis	18	80	60	Sodium cacodylate, 12 grains four times a day
11/ 9/21	5.1	50	75	Abductor pollicis brevis	7	104	60	
11/ 9/21	5.2	40	60	Gastrocnemius	36	104	60	
12/ 7/21	5.3	40	60	Flexor carpi radialis	28	...	60	No medication for 7 preceding days
12/22/21	5.8	..	..	Flexor carpi radialis	28	...	60	No medication for 3 preceding weeks
12/22/21	5.5	40	60	Flexor carpi radialis	28	...	60	1½ hours after scopolamin hydrobromid 1/100 grain by mouth
1/25/22	5.7	35	65	Flexor carpi radialis	28	88	61	Scopolamin hydrobromid 1/100 grain three times a day from December 22 to January 21
1/25/22	5.4	..	70	Flexor carpi radialis	28	88	61	
1/25/22	5.3	35	60	Flexor carpi radialis	28	88	61	½ hour after fluid extract gelsemium, 7 drops by mouth
2/ 8/22	5.6	35	60	Flexor carpi radialis	28	...	61	Fluid extract gelsemium, 4 drops three times a day from January 25 to February 8
3/29/22	5.2	40	65	Flexor carpi radialis	28	76	61	No medication
3/29/22	0	0	55	Flexor carpi radialis	28	82	61	5 minutes after scopolamin hydrobromid, 1/250 grain intravenously
3/29/22	0	0	45	Flexor carpi radialis	28	...	61	10 minutes after scopolamin as above
Average.....	.....	.....	62					

of a simple reflex such as the knee jerk,[4] which shows a single diphasic action-current. It is, however, quite similar to the contraction of clonus [12] except for the fact that between contractions there is a series of small waves (discussed in the foregoing) which possibly are due to the steady muscular rigidity.

Then there is the fact of the remarkable regularity of this tremor. In any one case the periods between tremor-contractions vary only a few hundredths of a second, and over long periods the tremor rates remain quite constant. For example, Case 3 (Table 2) had records made from the flexor carpi radialis in four successive months. On December 7 the rate of tremor was 5.3 per second; on December 22 it was 5.5; on January 25, 5.4; on February 8, 5.6, and on March 29, 5.2. Another patient (Case 13) on January 29, 1920, showed a rate of tremor in the flexor carpi radialis of 6.4 per second; twenty-two months later the rate was 6.5, and a month after this 6 per second. Other examples of regularity over varying periods may be found in Table 1. In fact it is difficult to vary the rate by any experimental or therapeutic procedures, although minor spontaneous changes occur within a few minutes. Drugs, for example, seem to have little effect on the tremor rate, although they may decrease the amplitude of the tremor and even cause its disappearance. Case 3 (Table 2) was studied from this point of view: In October, while in the wards of the hospital, the patient was given sodium cacodylate in large doses intramuscularly. This seemed to relieve the pain in his shoulder and decreased the muscular rigidity slightly,[13] but had no effect on the rate of tremor. Medication was then omitted until December 22 when scopolamin hydrobromid, $\frac{1}{100}$ grains, was given by mouth. This perhaps reduced the rate from 5.8 to 5.5, but the psychologic factor of waiting for the effect of the drug for one and one-half hours in a quiet room must not be forgotten. On March 29 the rate was 5.2 and the excursion ample (Plate 3, Fig. 1); five minutes after the intravenous administration of $\frac{1}{250}$ of a grain of scopolamin hydrobromid the electromyogram shows no trace of the tremor (Plate 3, Fig. 2), and clinically it appeared to have ceased. Five minutes later (Plate 3, Fig. 3) the tremor was still absent, and although slight tremor of the thumb was observed within half an hour, the patient said that he was comfortable and practically free from the "shaking" for eight hours.

The long waves so conspicuous in Plate 3, Figure 1, are not due to action-currents, but to shifting of the electrode on the skin of the patient, thus causing a change of the base line in the myogram. This

12. Footnotes 4 and 5.

13. Rodriguez, M. B.: Rev. neurol. **28**:699, 1921. Mella, H.: A Preliminary Report on the Treatment of Paralysis Agitans, Arch. Neurol. & Psychiat. **7**: 137 (Jan.) 1922.

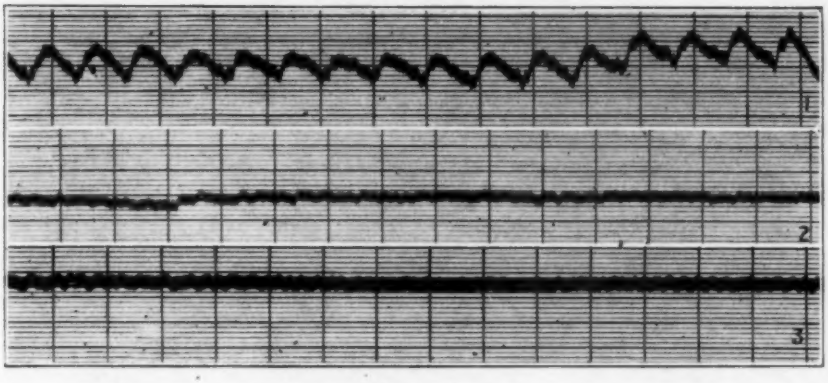

PLATE 3

Paralysis agitans, Case 3. Time marker, ⅕ of a second.

Fig. 1.—Myogram before medication. Rate, 5.2; marked tremor. The hand is unrestrained and there is shifting of the leads on the skin.

Fig. 2.—Five minutes after scopolamin hydrobromid, grains 1/250 intravenously. No tremor visible clinically.

Fig. 3.—About ten minutes after medication; no tremor visible. Twenty minutes after the medication slight intermittent tremor appeared, but the patient reports that he was practically free until eight hours later.

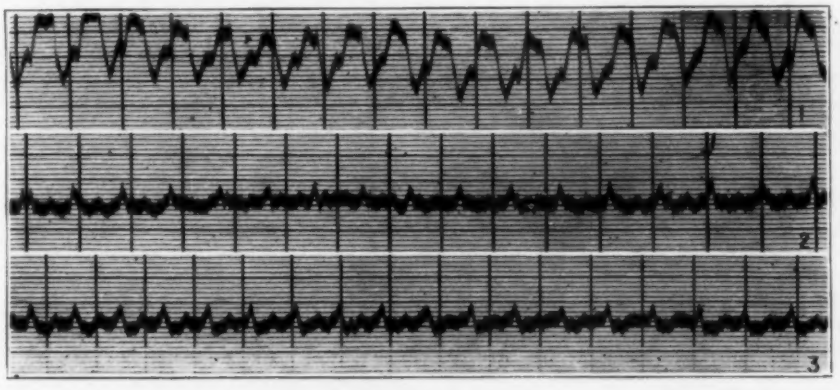

PLATE 4

Paralysis agitans, Case 3. Time marker, ⅕ of a second.

Fig. 1.—One-half hour after 7 drops of fluid extract of gelsemium had been administered. The motion of the hand was unrestricted so that there is marked shifting of the leads on the skin; rate, 5.7.

Fig. 2.—Same as Figure 1 but with restriction of the tremor by lightly restraining the wrist and hand; rate, 5.4.

Fig. 3.—After taking fluid extract of gelsemium, 4 drops three times a day, for two weeks; rate, 5.6.

is well illustrated in Plate 4, in which Figure 1 shows conspicuously the shifts in the base line; but Plate 4, Figure 2, after restriction of the mechanical fling of the hand caused by the tremor, shows no such shifting of the base line because the electrodes are stopped from shifting their positions on the skin. The tremor, however, continues to register through the electrical activity in the periodically contracting muscle.

Fluid extract of gelsemium seemed to have no effect on the myograms either when given over a long period (Plate 4, Fig. 3) or by making records immediately after a larger dose, as on January 25, 1922, in Case 3 (Table 2).

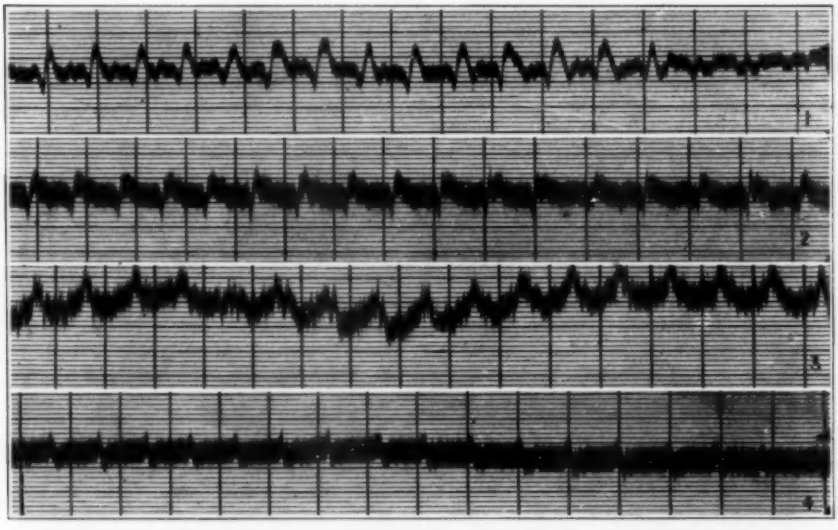

PLATE 5

Paralysis agitans, Case 3. Time marker, ⅕ of a second.

Fig. 1.—Forearm flexors; Dec. 7, 1921; rate, 5.3.
Fig. 2.—Gastrocnemius; Nov. 9, 1921; rate, 5.2.
Fig. 3.—Abductor pollicis brevis; Nov. 9, 1921; rate, 5.1.
Fig. 4.—Extensor pollicis brevis; Oct. 29, 1921; rate, 5.2.

A study of the data obtained from Case 3 indicates that the rate of tremor is practically the same in different muscles (Plate 5). In the beginning of this work we expected that short muscles might show a more rapid rate than long muscles, but the observations seem to prove conclusively that the rate of tremor is not a function of the length of the muscle. For example, Case 3 shows a rate of 5.1 in the abductor pollicis brevis (Plate 5, Fig. 3), a muscle approximately 7 cm. in length, whereas the gastrocnemius in the same patient on the same date had a rate of 5.2 per second (Plate 5, Fig. 2). The gastrocnemius is

approximately five times as long as the abductor pollicis brevis. Other muscles are shown in this same table with their approximate lengths and rates of tremor.

What, then, is the physiologic process that controls this so regular rate of periodic muscular contraction? It seems that a neuromuscular rhythm of such constancy must depend on some fundamental bodily condition. To obtain evidence on this point the heart rate in many of the patients was recorded at the same time that the myogram was made, but no correlation could be made out between these two rates, except the generalization that in young people in whom the heart rate (and the metabolic processes) are more rapid, the rate of the tremor is more rapid (Case 12, Table 1). This seems also to be true of clonus [12] and continues the interesting comparison between the two conditions.

The average rate of tremor for all the adult cases was found to be 5.8, whereas the boy of 8 (Case 12) had a much greater rate of 9 per second. Case 5 is not included because an Italian girl of 12 may be considered (metabolically speaking) on the borderline between childhood and adult life. The typical parkinsonian cases had an average rate of 5.9, whereas the atypical cases averaged 5.7. The males averaged 5.76 in rate and the females 5.77.

The basal metabolism was studied in Cases 9, 10 and 18. All were above normal, but this may be due to the muscular activity of the tremor. The question as to whether the rigidity of the muscles causes an increase in metabolism takes us again into the study of muscle tone and the various points touched on in the foregoing. Roaf [14] has found no increase in metabolism in a cat made rigid by decerebration.

If then this rate of tremor is so constant, and is independent of muscle length, it would seem to be a rhythmic discharge of the central nervous system. The fact that children show a more rapid tremor than adults indicates that general metabolism controls the rate. It is hoped that further research will elucidate these relationships, possibly explaining nervous discharge of this sort in some such way as Lucas and Adrian [15] have explained nerve conduction.

CONCLUSIONS

1. The tremor of paralysis agitans gives a characteristic electromyogram, with large, slow waves at the time of muscular contraction, and smaller, more frequent waves between these tremor-contractions.

2. The rate of the tremor of paralysis agitans is remarkably constant, the average being 5.8 per second. Little variation is observed in any one case when reexamined months later.

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14. Roaf, H. E.: Quart. J. Exper. Physiol. **5**:31, 1912.

15. Lucas and Adrian: The Conduction of the Nervous Impulse, London, 1917.

3. In children the rate of the tremor may be much more rapid (9 per second in Case 12).

4. Scopolamin may stop the tremor, but does not seem to slow the rate when acting less completely.

5. Various muscles in the same person show practically the same rate of tremor.

A CONSIDERATION OF THE DERMAL VERSUS THE EPIDERMAL CHOLESTEATOMAS HAVING THEIR ATTACHMENT IN THE CEREBRAL ENVELOPES *

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In a series of approximately 750 verified brain tumors from the clinic of Dr. Harvey Cushing, there have occurred seven examples of true intracranial cholesteatomas. In 1920, Bailey [1] reported two of these, representing the nonhair-containing variety, and since the publication of this article there has been one further example of the same type. The other three tumors have contained hair, and therefore come under the heading of dermoids, or hair-containing cholesteatomas. It is with the latter three cases that this paper proposes to deal.

NOMENCLATURE

A word should be said regarding the terminology used in the description of the tumors under discussion. By the term "intracranial cholesteatomas" is understood the definite but rare class of tumors of the intracranial cavity ordinarily referred to as "cholesteatomas" and qualified as "hair-containing" or as "pearly tumors." Twenty-five years ago Bostroem [2] (1897) gave an admirable description of these growths under the caption of "pial epidermoids and dermoids, and dural dermoids," using these terms because they represented what he believed to be their supporting tissue.

The term "pearly tumor" describes only a limited class of cholesteatomas, meaning merely that some tumors of this general group exhibit a definite pearly luster owing to the highly refractile quality of their delicate covering. This name has been applied heretofore to the nonhair-containing epidermoid variety, because it is that type which shows this peculiarity most strikingly, the visibility and pearly sheen usually being dependent on whether only the upper, and not the lower or dermal layer was included in the tissue of origin. That this distinction is not always true is shown by Bostroem's second case and by one

* Read at the Forty-Eighth Annual Meeting of the American Neurological Association, May, 1922, Washington, D. C.

1. Bailey: Cruveillier's tumeurs perlées, Surg., Gynec. & Obst. **31**:390-401 (Oct.) 1920.

2. Bostroem: Ueber die pialen Epidermoide, Dermoide und Lipome, und duralen Dermoide, Centralbl. f. allg. Path. u. path. Anat. **8**:1-98, 1897.

of the cases reported here, in which although the growths were in appearance "pearly tumors," nevertheless they contained hair. So far as can be learned, these are the only examples of such a finding, and bear out the almost certain general relationship of these tumors. That deeper layers may be involved, or at least that the cell inclusions may have even further potentiality, is shown by the occasional reports of certain intracranial teratomas, which are unquestionably only a step further and quite analogous to the growths under discussion (Kato).[3] Indeed one of our cases, as will be shown later, contained a small osteoid fragment and therefore might almost come under the latter designation.

Tumors of the hypophysis, arising from the remains of Rathke's pouch, sometimes reported as intracranial cholesteatomas because they contain cholesterin crystals, should not be included in our use of the term. They are not analogous tumors, either in respect to their tissue of origin or their general content. Likewise I do not consider the so-called "middle-ear cholesteatomas," since it is still a mooted question as to whether they are originally meningeal growths which have broken down and made their way to the surface, or whether they result simply from collections of the products of inflammation due to a chronic otitis media. A recent discussion of this subject was published by Crone,[4] in 1917, in which he reported six cases of "dermoid fistula of the temporal bone." That true intracranial cholesteatomas may occasionally occur in this situation and resemble the more commonly reported otitic collections is probably true. Grossman,[5] in 1903, found one or possibly two examples of this variety of tumor out of 578 cases of cholesteatomas from the Berlin Königlichen Ohrenklinik.

HISTORICAL NOTE

The story of meningeal cholesteatomas has been recited by almost every one who has reported tumors of this type, partly because their rarity and striking physical characteristics have warranted something more than a brief description of their pathology, and partly also because the confusion over the designation of their name has needed an historical explanation. The dermoid variety was described much earlier in the literature than the epidermoid. As noted by Bostroem, such a tumor was mentioned by Verattus[6] in 1745, which is sixty-two years

3. Kato: Ein kasuistischer Beitrag zur Kenntnis von teratoiden Geschwülsten in Kleinhirnbrückenwinkel, Jahrb. f. Psychiat. **35**:43, 1914.

4. Crone: Die Dermoid fisteln über dem Steissbein, München. med. Wchnschr. **16**:521, 1917.

5. Grossman: Ein ungewöhnlichen Befund bei Cholesteatom und Sinusthrombose, Deutsch. med. Wchnschr., 1903, No. 24.

6. Verattus: De Bononiensi scientiarum et artium instituto atque academia commentarii **2**: Pt. 1. 184, 1745.

prior to Dumeril's case[7] of the nonhair-containing type, reported in 1807, and depicted in 1829 by Cruveillier.[8] The latter investigator introduced the term "tumeur perlée," but in 1838 Johannes Müller,[9] referring to these cases of Cruveillier, in a treatise in which he also described two similar tumors which he had himself observed, called them cholesteatomas because they were found to contain cholesterin crystals.

The next important landmark in their history was the reversion to Cruveillier's terminology by Virchow,[10] in 1855, in his studies "Ueber Perlgeschwülste." As cholesterin could not be demonstrated in all cases of undoubted pearly tumors, the broadly descriptive term seemed more accurate. It was not until 1897 that an attempt was made to supply histologically exact designations for meningeal growths hitherto referred to as pearly tumors or cholesteatomas. In this year Bostroem, as mentioned previously, reported both a hair-containing and a nonhair-containing tumor of the type under consideration. By a most careful examination of the gross relationship to the cerebral membranes of the tumors which he reported, and further by exhaustive microscopic studies, Bostroem showed their definite attachment to the pia mater. In the dermoid which he presented, serial sections of the tumor's point of attachment demonstrated that its intrinsic elements, connective tissue, elastic fibers, blood vessels, etc., were continuous with those of the pia, and that its blood supply came from vessels which formed the direct supply of this membrane, with which they were continuous. As a result of these studies, he furnished the new appellations of "pial dermoids and epidermoids, and dural dermoids." These terms, however, are cumbersome, and have not gained recognition by subsequent writers, so that at the present time the most useful name, and the one which seems to be most universally associated with this variety of tumor, is cholesteatoma. If we now modify this very general term, and refer to tumors of this group as meningeal cholesteatomas, there can be no mistaking the kind of growth to which reference is made, as this designation immediately eliminates the group of tumors containing cholesterin crystals which arise from pituitary rests, and also does away with any confusion of association with those of the middle-ear cholesteatomas, which have not had a primary meningeal attachment. The occurrence of cholesteatomas, either hair-containing or otherwise,

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7. Dumeril: Bull. Soc. Faculté de méd., Feb. 19, 1807.

8. Cruveillier: Anatomie pathologique du corps humaine **1**: book 2, plate 6.

9. Müller: Ueber den feineren Bau und die Formen der krankhaften Geschwülste, 1838.

10. Virchow: Ueber Perlgeschwülste, Arch. f. path. Anat. **8**:371-418, 1855.

within any of the cerebral envelopes, from the scalp inward, has of course long been recognized. Some of these tumors, although not present in the meninges, may still be intracranial, as shown by a recent case from this clinic reported by Dr. Cushing.[11] Our use of the term meningeal cholesteatomas refers simply to a particular subgroup in the general class, in order to distinguish them from tumors which are not analogous.

INCIDENCE

Examples of the dermoid variety have been less commonly reported than those of the epidermoid. In Bostroem's exhaustive article in 1897 only eighteen reports of the former could be collected, while since that date we have found five further cases in an extensive search of the literature. These are contained in the articles of Tannenhain [12] (1897); Trachtenberg [13] (1898); Schulgin [14] (1911); Teutschlaender [15] (1914); and Stanojevits [16] (1918). Trachtenberg's case is of especial interest as it represents a rare example of multiple tumors, one of which was in the spinal canal, while the others were intracranial.

The eighteen cases found by Bostroem, together with the five just noted, make twenty-three cases reported up to the present time. These, with the three which I shall cite, make a total of twenty-six. Of the epidermoids, Bailey accounted for sixty-two in 1920, making that type appear to be more than twice as common. This relationship, however, is not borne out in the Brigham series, where, of 750 verified intracranial tumors, there have occurred three tumors each of the dermoid and epidermoid variety. This makes either type represent 0.4 per cent. of all intracranial growths.

LOCATION

The favorite situations of the dermal and epidermal cholesteatomas are very similar, their most frequent sites being somewhere near the midline at the base of the brain, or in the region of the fourth ventricle,

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11. Cushing, H.: A Large Epidermal Cholesteatoma of the Parieto-Temporal Region Deforming the Left Hemisphere Without Cerebral Symptoms, Surg., Gynec. & Obst. **34**:557-566, 1922.

12. Tannenhain: Dermoid Cyste des dritten Gehirnventrikels, Wien. klin. Wchnschr., 1897, p. 494.

13. Trachtenberg: Ein Beitrag zur Lehre von den arachnoidealen Epidermoiden und Dermoiden des Hirns und Rückenmarks, Arch. f. path. Anat. **154**:274-291, 1898.

14. Schulgin: Zwei Fälle von Cholesteatom des 4 Ventrikels, Sowrem. Psichiat., Moscow, 1911, No. 1, p. 143.

15. Teutschlaender: Zwei seltenere tumorartige Bildungen der Gehirnbasis, Arch. f. path. Anat. **218**:224-248, 1914.

16. Stanojevits, L.: Mannfaustgrosses, lange Zeit hindurch ohne objective Symptome bestehendes und plötzlich zum Tode führendes Klein hirnteratom, Neurol. Centralbl. **37**:784, 1918.

though in addition the dermoids have a strong predilection to push into one or other of the cerebral hemispheres, where they are usually much larger than the epidermal variety. The one striking point of difference, however, is that the cerebellopontile angle is one of the most common places for the nonhair-containing tumors, whereas no dermoids have been reported in this location.

Of the three cases from this clinic, one occurred in the middle fossa, compressing the temporal lobe. This situation has been reported only twice previously, and in each of these instances, as was true also in our case, the tumors were composed of two portions, like an hour-glass, as shown by the illustrations from the original articles. A summary of each of these reports is of interest.

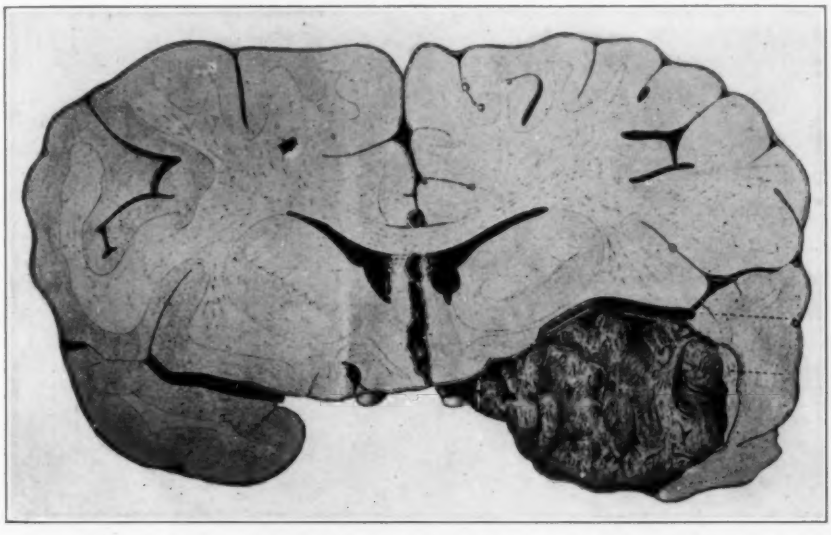


Fig. 1 (Bostroem's Case).—Transverse section of brain, showing tumor compressing left temporal lobe.

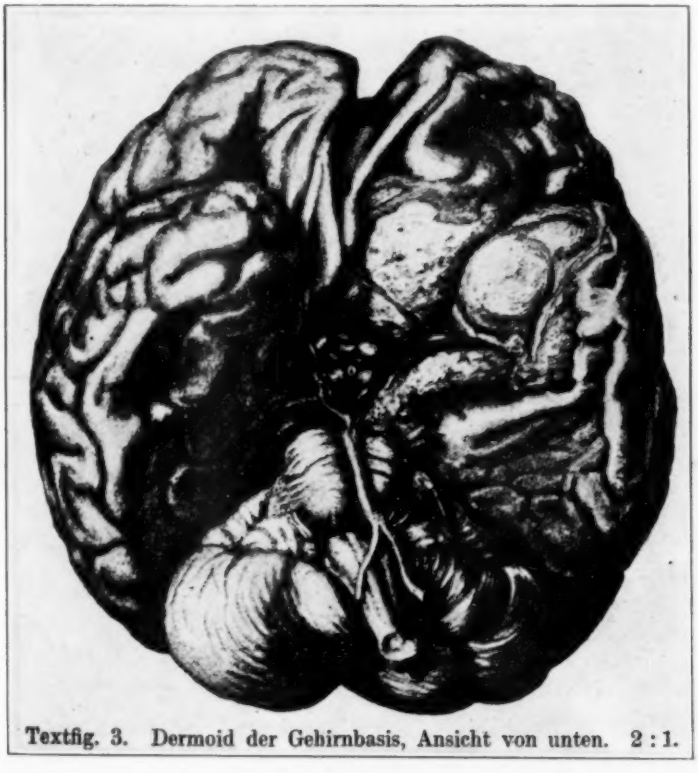
CASES IN THE LITERATURE

Bostroem's Case.—A man, aged 20, died in the workhouse, April 13, 1894, of extensive lung tuberculosis. While an inmate of the institution he had been lazy, obstinate and aggressive. His mental attainments were limited, and he complained frequently of sleeplessness. His left eye was reddened and swollen.

Necropsy revealed a tumor in the left fossa of Sylvius, compressing the temporal lobe (Fig. 1). The tumor was of a cloudy, opaque, dry consistency, yellowish green in color, with an exquisite pearl-like sheen over its surface. It measured 4.4 by 3.3 cm. in two diameters. It was covered by a delicate membrane which was continuous with the arachnoid. The tumor was further seen to consist of two portions, a larger lateral portion, and a smaller medial portion, the part between being compressed by the left ala orbitalis.

Section of the tumor showed a lumpy, yellowish-white, cloudy, opaque mass, with a firmer framework visible below. Shining white, pearly masses were present throughout the section, and between these were mingled large and small collections of soft, sebaceous material of a cloudy yellow color in which were many fine, short hairs, arranged in groups, mostly in the middle part of the tumor.

Sections of the tumor, both fresh and after embedding, showed a covering of epidermal cells, not so typically and plainly formed as in epidermoids. The contents consisted of thin, transparent, polygonal cells, most of them without nuclei. Between these cells there were masses of free, shining fat, and in the midst of this were many short, yellowish hairs and some cholesterin crystals.



Textfig. 3. Dermoid der Gehirnbasis, Ansicht von unten. 2 : 1.

Fig. 2 (Teutschlaender's Case).—Basal view, showing situation of tumor.

Teutschlaender's Case.—A 50-year old inmate of an insane asylum, a well educated man, had been subject to epileptic seizures for many years. During the last few months of life he was troubled with disorientation and periods of confusion. He became untidy. Death occurred Jan. 13, 1912.

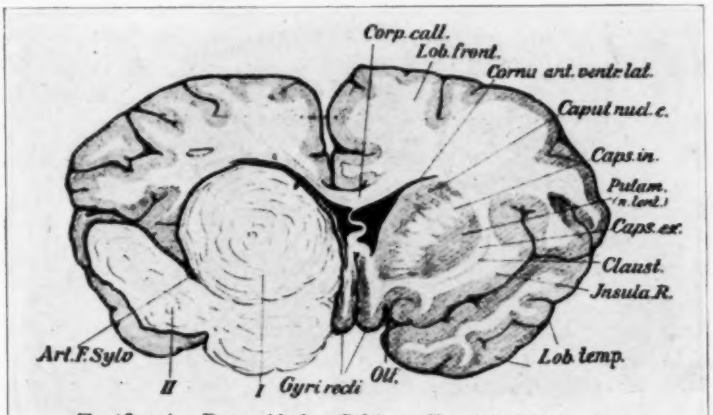
At the base of the left cerebral hemisphere, in the region of the fossa of Sylvius and the neighboring portion of the frontal and temporal lobes, there was found, at necropsy, a sebaceous, yellowish white, soft mass, the size of a goose egg, partly free and partly buried in brain substance (Fig. 2). The mass was separated from the brain by the pia-arachnoid. It consisted of two

portions, joined by a connecting bridge of tumor tissue. The larger medial portion was situated in the frontal lobe, and the smaller, lateral extension was in the temporal lobe (Fig. 3).

Gross transverse section showed crescentic layers of sebaceous material. The medial portion of the tumor contained macroscopic hairs. Chemical examination showed cholesterin.

Microscopic study demonstrated pavement epithelial covering, and on the inside fatlike material, hairs like lanugo hairs, and sebaceous glands.

These two findings are almost the exact counterparts of those in the first case which I shall report, and if a localizing diagnosis could have been made, they would have been surgically accessible. They illustrate, however, that only too frequent class of patients, who, for



Textfig. 4. Dermoid des Gehirns, Frontalschnitt. 2 : 1.

Fig. 3 (Teutschlaender's Case).—Transverse section, showing the large deeper portion and the small lateral extension of the growth.

one reason or another, because of ill-explained mental symptoms, become stranded in institutions for the insane, where necropsy reveals the cause of their disorder.

REPORT OF CASES

CASE 1.—*Hair-containing cholesteatoma of left temporal lobe disclosed by shadow in roentgenogram. Operation: extirpation of tumor. Recovery.*

History.—D. M. D., a man, aged 23 years, an army officer, was admitted Jan. 17, 1921, referred by Dr. Charles D. Humes of Indianapolis. He complained of headaches and convulsions. His family history was unimportant. He had had chorea in 1907; tuberculous adenitis in 1916, with excision of the glands in the left cervical region in 1917; a nervous breakdown in 1918; meningitis in 1919, and four operations for removal of the tonsils, the last one in 1918. In 1915, while running on an indoor track, he fell and struck his head on a post. He was dazed, and had a large scalp wound of the left frontal region.

Present Illness.—After an illness called a "nervous breakdown," in 1918, the patient dragged his left leg for about a month. He recovered from this and was in active service with the United States Army in France from July to November, 1919. In December, 1919, he had a peculiar seizure in a hotel. He was seated at a table, when quite unaccountably, he began to act peculiarly, and upset a glass of water in an awkward fashion. He did not answer questions. He excused himself from the table, and while in the hotel lobby became dazed and then had a general convulsion with vomiting. He was unconscious a large part of the time for two days, and had fever up to 103 F. for four days. A diagnosis of influenzal meningitis was made. For three weeks after this seizure the patient had difficulty in speaking. He could not think of the words he wished to say and would have to stop in the middle of a sentence because he could not remember the proper word. He was disoriented and felt dull. He was up and about after three weeks, but for the next five months he had a good deal of pain in the back.

In January, 1920, he had an attack of vomiting, and fainted. In July, 1920, he had another convulsion with loss of consciousness lasting two hours. Roentgenograms of the skull taken after this seizure showed calcium deposits in the left temporal region. The spinal fluid examination was negative.

From this time until admitted to the Peter Bent Brigham Hospital, the patient had one convulsion, in September, 1920. He felt weak and without ambition, and became extremely tired on the slightest exertion.

Headaches localized in the left frontal region and associated with dizziness were present every day or two. During these attacks his pulse rate was very slow, sometimes less than 50 per minute.

Physical Examination.—Physical examination revealed almost nothing of significance. The left patellar reflex was somewhat greater than the right. Ophthalmoscopic examination of both fundi revealed rather full vessels, otherwise the eyegrounds were normal. There was slight fulness and tenderness over the left temporal region, and the vessels over this area pulsated more fully than on the right side. The visual fields were normal even to very small visual angles.

A convulsive seizure was witnessed on Jan. 18, 1921. This was preceded by a short period of mental dulness, amnesia and speech difficulty, especially inability to name objects. The convulsion itself consisted in an initial drawing of the head to the right, followed by twitchings of both sides of the face, and finally by clonic jerkings of first the right arm and right leg, and then of the entire musculature on both sides of body.

Stereoscopic plates of the skull in a left lateral position showed a thin, crescent-shaped shadow of increased density, apparently above and to the left of the sella turcica (Fig. 4). The Wassermann test of the blood and spinal fluid were negative. Blood pressure, blood count, blood smears and urine were normal.

Operation.—Feb. 3, 1921, Dr. Cushing made an osteoplastic exploration of the left hemisphere. A temporal lobe cyst was extirpated. Ether anesthesia was used.

An osteoplastic flap was turned down with its base low in the left temporal region. The bone flap showed evidence of pressure erosion, the dura was quite tense and a subtemporal bone defect was made.

The dura was opened over the temporal lobe which protruded markedly, palpation showing what was unmistakably a cyst. An incision through the

second temporal was carried down to the wall of the cyst, and its upper pole was fixed with pledgets of Zenker's solution until it was stiff enough to handle. A small needle was then inserted into the cyst, but through this nothing could be withdrawn by suction. The usual brain needle was then inserted and a curious, grumous, dirty fluid was removed, immediate examination of which showed fatty cells. The remainder of the fluid as it adhered to the tube became

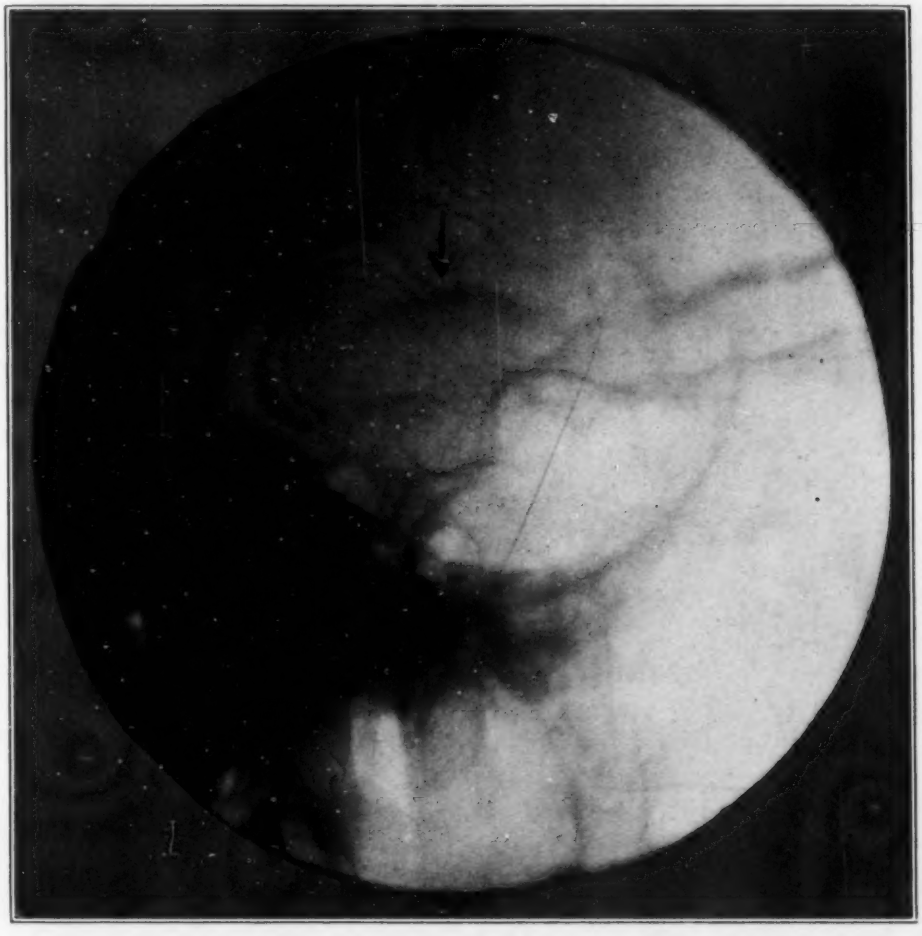


Fig. 4.—Crescentic shadow above the sella turcica, shown by the roentgenogram in Case 1.

stiffened, much like the cooling of melted fat. The fluid was thick and brownish, and about 40 c.c. were collected, although the cyst probably contained twice that amount as much of it was lost and the rest evacuated subsequently.

The cyst when emptied was found to have a delicate vascularized lining membrane. A curious fragment of tissue, quite loose, saucer shaped, about 1 cm. in diameter and 0.5 cm. in thickness, was lifted out of the cavity, the source of this fragment not being apparent. The cyst wall was carefully fixed

with Zenker's fluid until it became stiffened and puckered, then by filling the cavity full of cotton, it was possible by slow dissection to remove the entire cyst wall, though in the process it was somewhat shredded. The bone flap was then replaced.

Microscopic Examination.—The cyst contained a dirty, brownish fluid which had the appearance of thin gravy, but which felt like machine oil, owing to the large amount of fat. Microscopically, the field was covered with fat droplets which stained with scharlach r. There were also a moderate number of large, pale cells containing lipoid, and rare red blood cells.

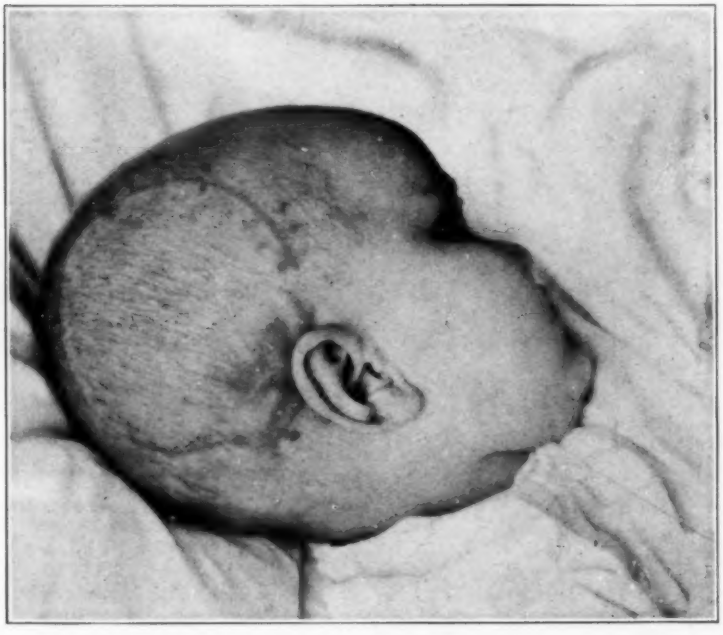


Fig. 5 (Case 2).—Patient at time of discharge from hospital.

Sections of the cyst wall showed that it was lined with a low, stratified epithelium, usually about 3 cells in thickness, but in places from 4 to 6 cells. Beneath this was a loose tissue in which were a moderate number of thin-walled blood vessels.

A section of the free body in the cyst cavity showed that it consisted of necrotic tissue débris in which were a great many fat crystals. A rare group of from ten to fifteen bodies which suggested epithelial scales was also seen, and rarely what was apparently a piece of hair cut transversely.

Diagnosis: Obscure cyst—cause unexplained.

Postoperative Notes.—Contrary to expectation, the patient did not do well. He remained drowsy and listless, and he complained frequently of headache. On February 25 his eyegrounds showed bilateral choked disks of 2 diopters,

whereas they had previously been normal. On several occasions he had a feeling of numbness in the right hand and arm.

On February 26, stereoscopic roentgenograms of the skull showed in addition to the operative defect the same crescentic shadow of increased density which was previously described.

On March 4, he had a generalized convulsion, and for the next ten days was drowsy with slowed pulse and respiration. The decompression area became full and tense, and on March 18 the elevation of the optic disks had risen to 3 diopters. It was evident that there was something wrong, and a reexploration of the wound was deemed advisable, particularly in view of the fact that the supposedly calcareous wall of the cyst had not been removed as the roentgen ray had demonstrated.

Second Operation.—March 21, 1921, Dr. Cushing made a subtemporal exploration through the previous operative site. He punctured the lateral ventricle. The second cyst, containing hair and débris, was evacuated.

A vertical linear incision was made through the temporal region, and the bone defect of the former operation enlarged. The dura was incised, disclosing an adherent temporal lobe which was protruding slightly. A needle was inserted into the ventricle, and 50 c.c. of clear fluid were secured.

In the lower portion of the temporal lobe there was disclosed the slightly fluctuant wall of what was taken to be a portion of the original cyst which had escaped notice at the primary operation. The enucleation of this was therefore undertaken, but during this operation an underlying cyst containing a considerable amount of granular, purulent material was encountered. The cystic cavity proved to be quite large and extended to the inner portion of the temporal lobe. In addition to the thick fluid evacuated, it contained much granular débris, in which were numerous soft, delicate hairs. Lining the cavity, moreover, there was a calcareous shell, which had evidently produced the shadow seen repeatedly in roentgenograms.

The wall of the cyst was cleaned and washed out with a weak formaldehyd solution; the wound was then closed, leaving a single drain into the depth of the cavity.

Pathology.—A considerable mass of the thick, granular contents of the cyst was hardened in Zenker's fluid. Sections of the contents showed numerous masses of epithelial scales, many minute pieces of hair, much fat and many polymorphonuclear leukocytes.

Diagnosis: An infected dermoid cyst.

After this procedure the patient made an uneventful and complete recovery. The drains were permanently removed twelve days after operation, and the wound was healed one week later. The choked disk receded to normal, and all of the patient's previous symptoms cleared up quickly. Further stereoscopic roentgenograms of the skull showed no shadow in the temporal region. The patient was discharged April 30, 1921, and in January, 1922, he reported that he was in excellent health.

Comment.—So extremely insignificant was the evidence of organic intracranial lesion in this patient that an exploratory operation was undertaken with great misgivings. The man had become a little dull and listless, three or four convulsions had taken place, and a roentgenogram examination of the skull had shown a faint shadow above the sella

turcica in the left hemisphere. In favor of a left-sided lesion was the history of transient speech disturbances and the location of his headaches to the left frontal region. On the contrary, he had had a period during which he dragged his left leg, and objectively the left knee reflex was greater than the right. Chief credit should be given to the patient's family physician for strongly urging an exploration. The tumor was evidently of the hour-glass variety, the more external portion of which, showing no definite dermal elements in the wall, was encountered and removed at the primary operation; the mesial portion, containing the calcareous shell and dermal contents, was disclosed and extirpated only after the patient's unsatisfactory convalescence had made a second operative exploration seem advisable.

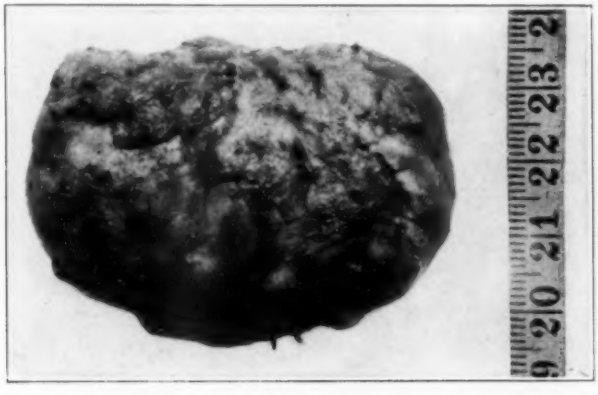


Fig. 6 (Case 2).—External surface of tumor.

CASE 2.—*Infected dermoid cyst of right temporal lobe. Two previous operations with disclosure of a small extradural abscess containing hair. Exploratory craniotomy. Removal of a thick-walled cyst from right temporal lobe. Temporary recovery. Death three and one-half months later.*

History.—M. L. W. was referred by Dr. St. Clair of Johnson City, Tenn., with a diagnosis of brain tumor. The patient, a child, 3½ years of age, was admitted to the hospital May 28, 1909. The family history was unimportant. The child was born at full term by normal labor. She was healthy and breast fed. At the age of 4 months she had evidently had an otitis media, with a discharge of pus from one of her ears. It could not be ascertained which ear was involved. She recovered quickly from this illness. When 8 months old she had "intestinal indigestion" lasting several months during the summer, and she had never been entirely well after this trouble.

Present Illness.—In September, 1908, the child was ill with a sore throat, and one month later an abscess on the right side of her neck was evacuated. About this time she began to have pain in the head, and a slight weakness of the left side of the face was noted. In December, 1908, a reddened, swollen area behind the right ear was incised, but no pus was obtained. Five days later

it was again incised and considerable pus evacuated. In January, 1909, the same area was incised and the bone curetted. By the end of January the child had developed weakness of the whole left side. The area behind her right ear was again opened, and this time an abscess containing hairs was disclosed between the bone and dura. The condition cleared up temporarily, but on February 26 it was again found to be necessary to open the wound, and again pus and hairs were disclosed between the bone and dura. Once more the symptoms cleared up for a time, but shortly before admission to the Johns Hopkins Hospital she had a left-sided convulsion, accompanied by headaches and vomiting.

Neurologic Examination.—The child was pale, irritable and restless. Percussion of the head elicited definite "cracked-pot" resonance. Her pupils were unequal, the right being slightly larger than the left. The right fundus showed a typical choked disk with an elevation of 2 diopters, while the left fundus

Fig. 7 (Case 2).—Inside of tumor.

was normal. There was slight weakness of the left leg, and the deep reflexes were more active on the left side. Ankle clonus was present on both sides. There was a tender, reddened area behind the right ear, with a discharging sinus.

Operation.—May 31, 1909, Dr. Cushing made an exploratory craniotomy and lumbar puncture disclosing a large cyst of the right temporal lobe. The cyst was extirpated and the child recovered.

A bone flap was turned down over the right temporal region, disclosing a tense dura which was adherent at the site of the previous operations. The dura was opened over the lower portion of the field, and there was considerable bulging of the brain through the opening, necessitating lumbar puncture. The appearance of the cortex suggested an underlying cyst; therefore an incision was made in the first temporal convolution, disclosing a cyst wall. This wall was dissected free from the surrounding brain and removed entirely, though in doing so it was necessary to open the cyst and evacuate its contents. The cavity contained a thick, greenish purulent material, cultures from which showed a pure strain of streptococcus.

The dura was closed as far as possible, the bone flap replaced and the wound sutured, leaving a single drain into the cavity from which the cyst had been extirpated.

Postoperative Notes.—The child made a good primary recovery, but there were wound complications which necessitated prolonged hospital care, and though discharged apparently well (Fig. 5), she died three and a half months after the operation.

Pathology.—Gross Appearance of Tumor: The tumor was roughly elliptical, about the size of a hen's egg, measuring 5.5 by 4 by 3 cm. in its three diameters. The surface was fairly smooth, but with irregular elevations and depressions (Fig. 6). A longitudinal section dividing the tumor from one end to the other showed that it was entirely cystic, with a tough, fibrous wall which varied from 3 mm. to 7 mm. in thickness. The entire inner surface of the cyst wall was trabeculated with interlacing cords, which in some places stood out as isolated bands but in other places merged into the general structure of the wall (Fig. 7).

Microscopic Description of Cyst Wall: The tissue consisted of a cyst wall, such as is seen in epithelial cysts, especially those of the dermoid variety, that is, a wall made up of fibrous tissue abundant in collagen. The cyst lining was made up wholly of granulation tissue in which were many large cells with small nuclei which contained fat vacuoles. They were evidently phagocytic cells of the endothelial variety. There was also a considerable infiltration of polymorphonuclear leukocytes and lymphoid cells. No hair follicles were discernible.

Diagnosis: Probable dermoid cyst (infected).

Comment.—The patient had been operated on two or three times previously, incisions having been made over the postauricular region into what was at first supposed to be a superficial abscess. At one of these sessions a trephine opening had been made in the skull, and subsequently, on another occasion, through this opening was disclosed what was taken to be an extradural abscess, but in this abscess an abundance of short yellow hairs were demonstrated. Finally, a thick-walled cyst from the temporal lobe was removed beneath the region which had heretofore been superficially dealt with. The cyst contents had become infected, and at the time of extirpation no hairs could be demonstrated, but studies of the pathology of the wall of the cyst, and also the fact that hairs had been found in its infected contents previously, left no reasonable doubt as to its dermoid character.

CASE 3.—*Extreme grade of internal hydrocephalus with intracranial pressure symptoms. Operation without disclosure of tumor. Necropsy: hair-containing cholesteatoma of undersurface of cerebellum, giving mother-of-pearl appearance.*

History.—E. C., a white, female child 2 years of age, admitted Jan. 31, 1920, was referred by Dr. Z. L. Shaw of Manchester, N. H. The complaint was: "Large head." The family history was unimportant. The child was born at full term without instruments. Her appearance at birth was normal, and she developed normally in every way up to the age of 1 year. When 15 months old she fell out of her carriage to the sidewalk, striking the back of her head. She cried and vomited once after this fall, but seemed to suffer no immediate effects otherwise.

Present Illness.—At the age of 1 year the mother had to increase the size of the child's bonnets more than usual, and she also thought that her head was a little larger. At 17 months projectile vomiting began, which continued until two months before admission. The size of her head increased rapidly after the age of 17 months. At the age of 23 months she started to decline, having "spasms" with opisthotonos and loss of consciousness. She had not been able to sit up for one month. There was an increase in the size of the head of 4 inches (10.16 cm.) from July, 1919, to January, 1920.

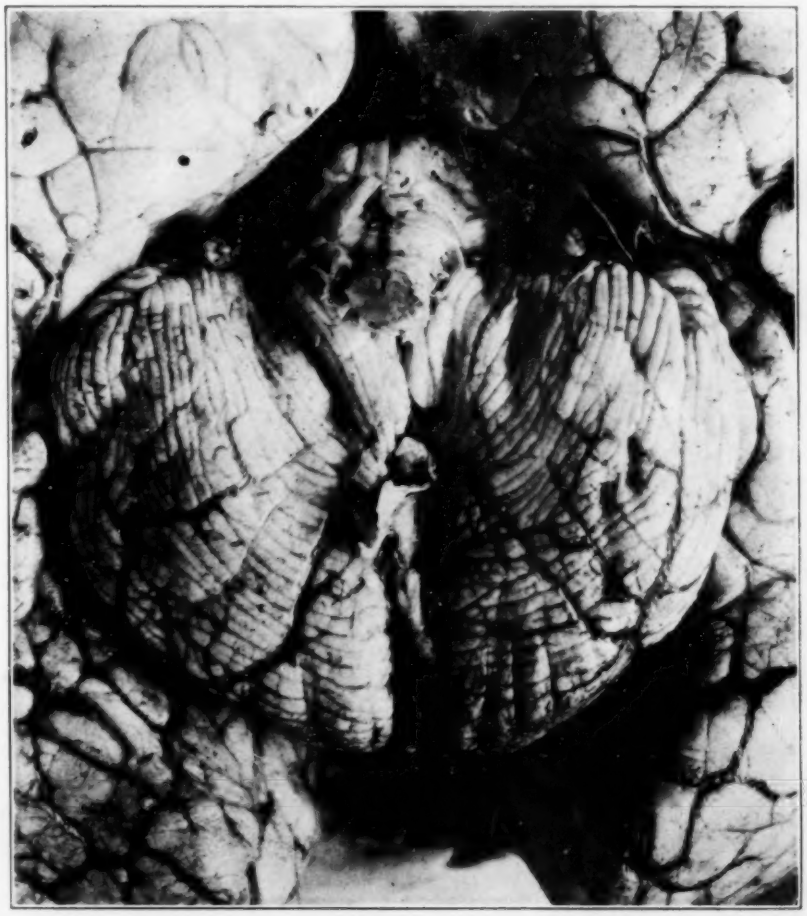


Fig. 8 (Case 3).—Inferior surface of cerebellum, showing, between the hemispheres, the area of thickened arachnoid membrane overlying the dermoid growth.

Physical Examination.—The patient was a well developed and well nourished child with evident hydrocephalus. The circumference of the head was 28 inches or 71.1 cm. There was marked separation of all sutures of the skull and great dilatation of the superficial veins over the scalp, with a marked bulging of the frontal region, and exophthalmos. The suboccipital region was full, especially on the right. She had a persistent lateral nystagmus to the right and left, and

also upward. There was definite incoordination of the hands. There was a large patch of craniotabes in the occipital region. In the midoccipital region, about at the torcular, there was a deep follicle from which several hairs grew like a pilonidal sinus. There was a huge median vein, 1 cm. in breadth, coming from the torcular. Examination of the fundi showed bilateral early choked disks of low elevation.

Operation.—On Feb. 3, 1920, Dr. Cushing made a suboccipital exploration for possible tumor of the cerebellum producing hydrocephalus.

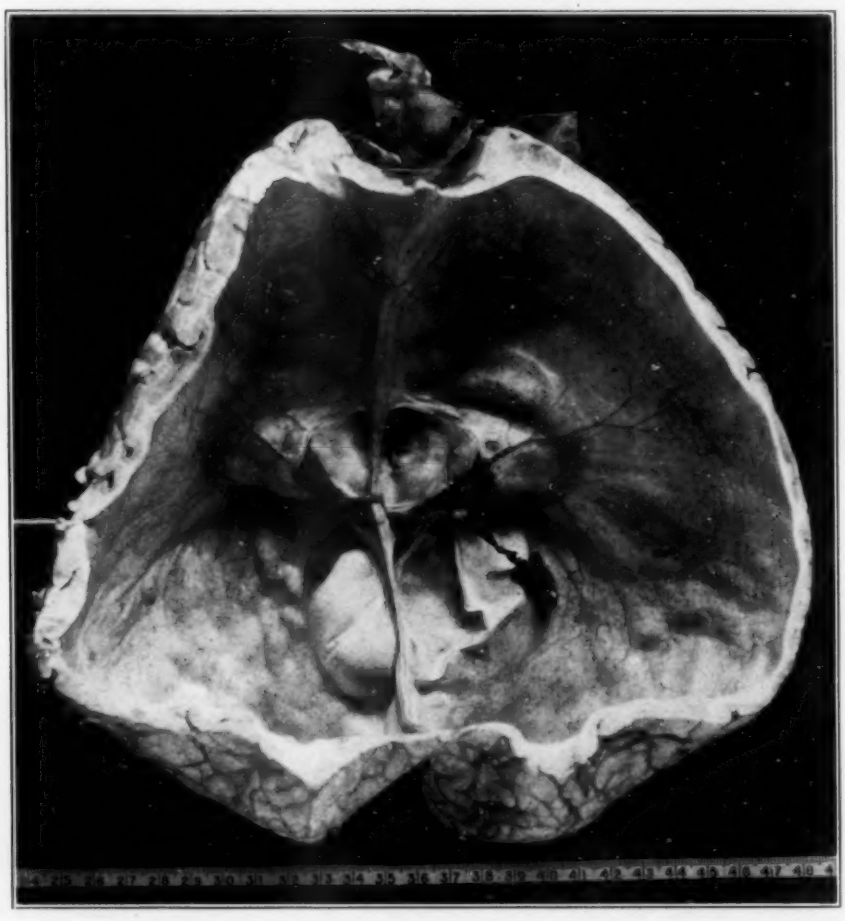


Fig. 9 (Case 3).—Interior of brain, showing extreme degree of internal hydrocephalus.

The suboccipital region was exposed by reflecting the skin and muscle flaps after the usual "crossbow" incision. Just above the operative area there was a tangle of dilated veins, radiating like a caput medusae from the torcular.

The occipital bone was exposed without great difficulty. It was not especially thin, nor was there any protruding of the suboccipital region. The dura, on exposure, was found to be tense, and a lumbar puncture needle was inserted

Fig. 10 (Case 3).—Longitudinal section of the tumor dividing the cerebellar hemispheres. Many long hairs may be seen growing into the tumor from the stalk at its posterior end.

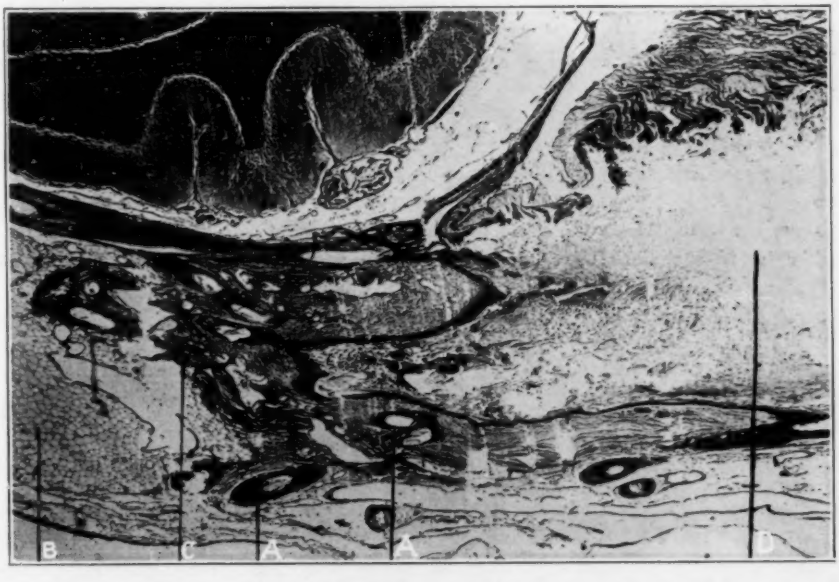


Fig. 11 (Case 3).—Photomicrograph taken through stalk of tumor, at point shown in Figure 14; *A*, hairs and hair follicles; *B*, fat cells; *C*, sebaceous gland cells; *D*, interior of the dermoid; × 25.

into each hemisphere in the hope of encountering a gliomatous cyst. Clear cerebrospinal fluid under tension was disclosed, and without opening the dura the operation was abandoned.

The child died six days after the operation of bronchopneumonia.

Necropsy.—The brain was fixed by injection of liquor formaldehydi through the carotids. The brain was enormous with extreme dilatation of the ventricles. The cortex was thin, measuring in some places only 3 mm., and was only about 6 mm. in its thickest portions. The dura was extremely delicate and thin. The cerebellum as compared with the rest of the brain was very small, measuring 9 cm. in its transverse diameter. Between the two cerebellar hemispheres there was thickened arachnoid overlying a small congenital tumor of the cholesteatomatous variety (Fig. 8). No other abnormalities were found except an extreme degree of internal hydrocephalus (Fig. 9) for which the small tumor to be described could in no definite way account.

Gross Description of Tumor.—Underneath the somewhat thickened arachnoid, between the inferior surfaces of the two cerebellar hemispheres a small oval tumor was disclosed, measuring 2.4 by 1 by 0.8 cm. in three diameters. It lay between the arachnoid and the pia, being everywhere separated from cerebellar substance by the latter membrane. The surface of the tumor was smooth and its wall translucent, showing plainly the white contents of the tumor, and giving much the same "mother-of-pearl" luster which has been used to describe the external appearance of the epidermoid cholesteatomas.

On longitudinal section, the tumor was seen to be surrounded by a thin, milky-white membrane which constituted its wall. This was slightly thicker than ordinary tissue paper and was fairly tough. The tumor was entirely filled with a white, rather granular substance, which had somewhat the appearance of cottage cheese. In this substance were numerous, fine, light-brown hairs, varying in length from 0.5 to 2 cm., and apparently growing into the white material from many places on the wall of the tumor (Fig. 10). There was one point at the posterior end of the tumor which seemed to represent its stalk, or spot of attachment apparently to the pia, and from this point hairs grew in greater abundance than at any other place.

Microscopic Description.[17]—The wall of the tumor was a thin membrane composed of two layers. The outer layer consisted of several strands of connective tissue, sometimes compact and sometimes loosely separated, in the meshes of which were occasional blood vessels, and in several places hairs. The inner layer was composed of cuboidal epithelium, in most places 1 or 2 cells deep, but in other places widening out to a depth of 8 or 10 cells.

At the posterior end of the tumor, the outer connective tissue layer of the wall was enormously widened into what appeared to be the stalk of the tumor where it had its attachment. In this stalk were numerous blood vessels, a great many hair follicles and many sebaceous gland cells surrounded by fat (Fig. 11). Most of the hair follicles pointed directly toward the interior of the tumor, evidently to discharge their hairs within it, although in addition to the hairs seen in cross section, inside the tumor there were others which had been cut across as they lay in the wall. At one point in the outer connective tissue layer a small area of osteoid tissue was present (Fig. 12).

17. For areas in tumor where microphotographs were taken, compare Figure 14.

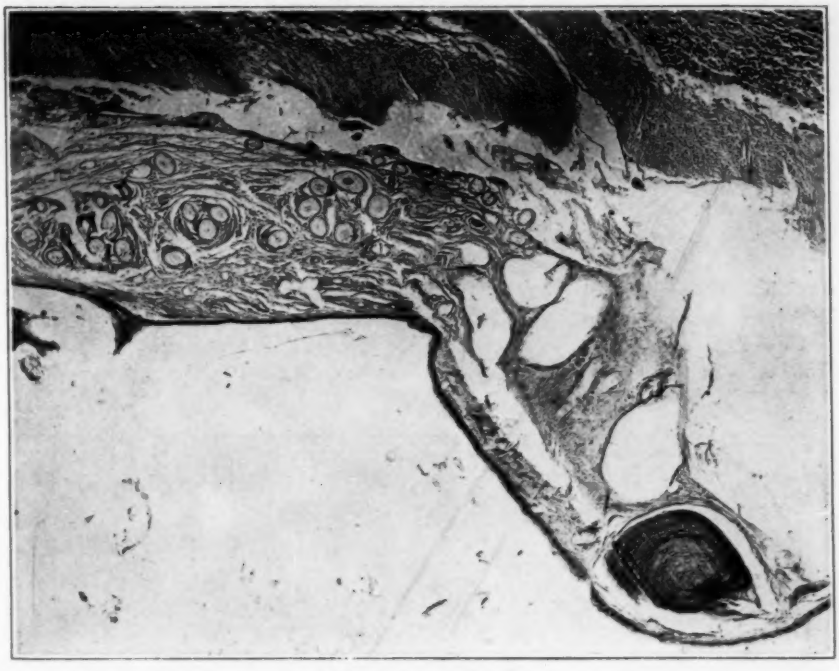


Fig. 12 (Case 3). — Photomicrograph taken at point shown in Figure 14. Osteoid tissue and hairs shown in wall of tumor; × 70.

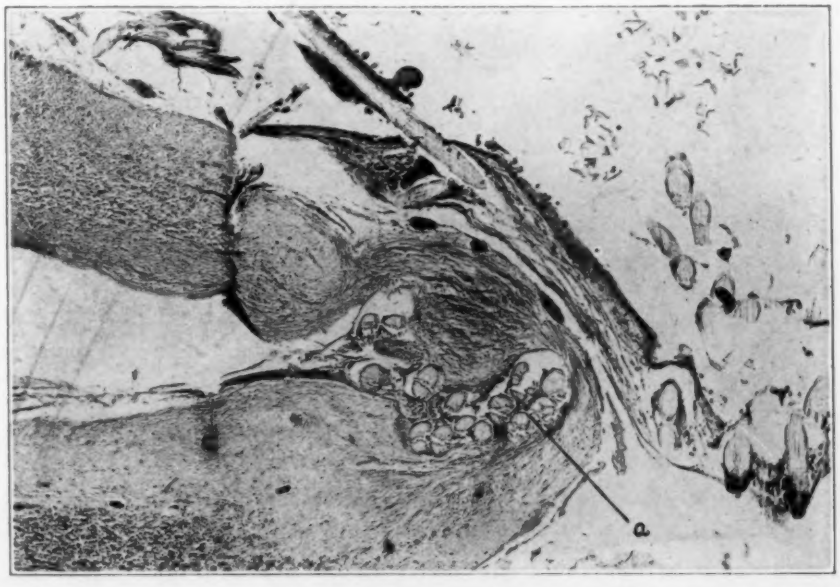


Fig. 13 (Case 3).—Photomicrograph taken at point shown in Figure 14, illustrating the hairs which have grown out into cerebellar substance at *a*; × 70.

The contents of the tumor could also be divided into two layers, the outer of which was represented by lines of homogeneous material without structure and without distinguishable cell boundaries. Below this layer, and making up the bulk of the tumor contents, was a mass of broken up, apparently disintegrating, polyhedral cells, the outlines of which were sometimes quite distinct, and whose nuclei could often be seen plainly.

In addition to the hairs seen in the tumor and within its walls, there were other groups of hairs cut transversely, which were entirely outside the tumor wall, lying in cerebellar substance (Fig. 13). The only explanation of the situation of these hairs appeared to be that they had grown out through the wall of the tumor, like "ingrowing hairs" in the skin, since the inside of the tumor would represent the outside of the skin.

Chemical analysis of the contents of the cyst disclosed the presence of cholesterin in considerable quantity, although no crystals could be demonstrated.

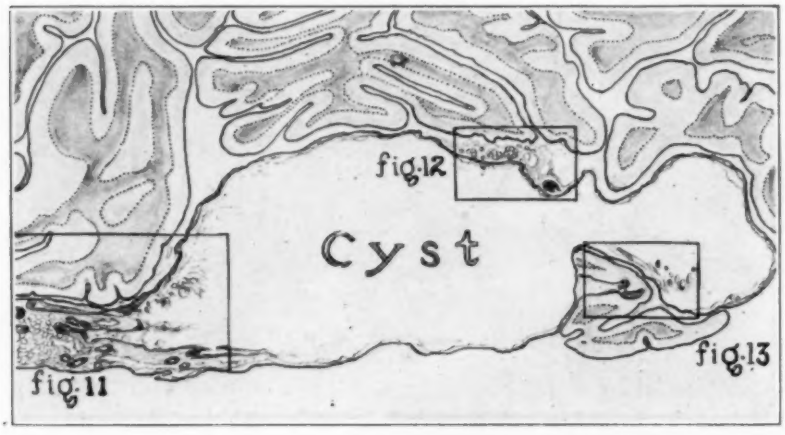


Fig. 14 (Case 3).—Diagram of tumor to illustrate areas from which photomicrographs were taken.

Comment.—From a pathologic and histogenetic point of view the tumor in this third case was undoubtedly the most interesting of the three here reported. It was, in effect, a pearly tumor, attached to the pia mater, its soft, white contents being somewhat similar to those of epidermoids, but with the addition of hair, while in the wall were hair follicles, sebaceous gland cells, fat and osteoid tissue. Its wall was, to be sure, somewhat thicker than in the nonhair-containing variety, since it probably represented an inclusion of deeper cell layers, but the wall was translucent, and there was a definite pearly sheen. Its median position at the base of the brain corresponded to one of the usual locations of intracranial cholesteatomas.

These striking points of similarity of a true dermoid to the nonhair-containing tumors of this class form a strong argument in favor of the congenital origin of the latter from superficial epiblastic cells which do

not have the potentiality of the lower layers. This bears out Bostroem's contention for the general relationship of this group of tumors, and further supports the views of others who have argued in favor of an epithelial origin for the nonhair-containing cholesteatomas.

SUMMARY

There is a group of rare tumors of the intracranial cavity which represent fetal epiblastic inclusions, sometimes of the epidermal layer alone, and sometimes including also the dermal layer.

These tumors may or may not contain hair and other tissue elements, according to the depth of the cell layer represented in the inclusion.

It is convenient to group all these tumors under the term cholesteatomas, either hair-containing or nonhair-containing.

Three examples of the hair-containing variety, or intracranial dermoids, are presented for consideration, and in two of the patients the tumors were removed by operation, in one of them with apparent success.

EPIDEMIC (LETHARGIC) ENCEPHALITIS

CULTURAL AND EXPERIMENTAL STUDIES. SECOND COMMUNICATION *

WILLIAM THALHIMER, M.D.
MILWAUKEE

The results of some experimental and cultural studies of epidemic encephalitis were reported in a preliminary communication [1] and were believed to confirm the demonstration by Loewe and Strauss [2] of a minute, filtrable organism as the cause of this disease. Additional material has been studied, and similar results have been obtained and are presented in this report.

Investigations by Levaditi and Harvier [3] and by McIntosh and Turnbull,[4] were referred to in the previous article.[1] Further studies by these investigators and reports by others, also confirming the experiments of Loewe and Strauss, have since been published, and it is of interest to review briefly some of this literature.

McIntosh [5] reported successful transmission from his original monkey to another, and to a rabbit. A monkey kept as a control with the inoculated monkey developed a spontaneous infection, with symptoms of epidemic encephalitis and characteristic brain lesions.

Levaditi, Harvier and their co-workers [6] have added more experimental evidence of the filtrability of the virus of epidemic encephalitis which is present in the central nervous system of fatal cases. They found it pathogenic for guinea-pigs and rabbits, but less so for monkeys. Animals were infected by inoculating them intracranially, intra-ocularly,

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* From the Laboratories of Columbia Hospital.

1. Thalhimer, W.: Epidemic (Lethargic) Encephalitis, Arch. Neurol. & Psychiat. **5**:113 (Feb.) 1921.

2. Loewe, L.; Hirshfeld, S., and Strauss, I.: Studies in Epidemic Encephalitis (Encephalitis Lethargica), J. Infect. Dis. **25**:378 (Nov.) 1919; New York M. J. **19**:772, 1919. Loewe, L., and Strauss, I.: Etiology of Epidemic (Lethargic) Encephalitis, J. A. M. A. **73**:1056 (Oct. 4) 1919; The Diagnosis of Epidemic Encephalitis, ibid. **74**:1373 (May 15) 1920; Proceedings of the New York Path. Soc. **20**:18, 1920; Studies in Epidemic (Lethargic) Encephalitis, Cultural Studies, J. Infect. Dis. **27**:250, 1920.

3. Levaditi, C., and Harvier, P.: Compt. rend. Soc. de biol., **83**:354, 1920; Bull. et mém. Soc. méd. d. hôp. de Paris **64**:179, 1920.

4. McIntosh, J., and Turnbull, H. M.: Experimental Transmission of Encephalitis Lethargica to a Monkey, Brit. J. Exper. Pathol. **1**:89, 1920.

5. McIntosh, J., and Turnbull, H. M.: The Experimental Transmission of Experimental Encephalitis Lethargica in Series in Monkeys and Rabbits, with Notes on a Spontaneous Infection in a Monkey, Brit. J. Exper. Path. **1**:257, 1920.

6. Levaditi, C., and Harvier, P.: Etude experimentale de l'encephalite dite lethargique, Ann. de l'Inst. Pasteur **34**:911, 1920.

into the peripheral nerves and intranasally after scarifying the nasal mucous membrane. They also found the virus in the cerebrospinal fluid and nasal washings of cases of this disease. Their attempts to cultivate an organism from the virus, with the methods perfected by Noguchi, were unsuccessful. The virus, however, remained alive in Noguchi medium for from seven to eight days and in tissue cultures, after the method of Carrel, for fifteen days.

Maggiore and Sindoni [7] isolated from the blood and spinal fluid of cases of epidemic encephalitis an organism resembling that isolated by Flexner and Noguchi [8] from cases of poliomyelitis. They produced the disease in rabbits by intracranial injection of a patient's spinal fluid and intravenous injection of a culture of the organism. Repeated passages through animals produced invariably the characteristic clinical picture and pathologic lesions. They concluded that poliomyelitis and epidemic encephalitis are identical. In a recent communication they report further cultural and clinical evidence which strengthens their belief that epidemic encephalitis and poliomyelitis are the same disease. (The presence of the virus in spinal fluid and the susceptibility of rabbits to the disease differentiate it from poliomyelitis.)

Ottolenghi, Antona and Tonietti [9] found the virus in the blood, nasal washings and spinal fluid of cases of epidemic encephalitis. The virus is filtrable. Guinea-pigs were infected by intracranial and intraperitoneal inoculation and by instillation of nasopharyngeal washings into the nostrils. In rabbits the nasal mucous membrane had first to be scarified for successful inoculation. Brains of cats, experimentally infected, were virulent for guinea-pigs. Two strains of virus were successfully passed through eight and twelve series of animals. The experimental disease lasted from five to thirty-five days and proved fatal in all but thirteen of the 215 guinea-pigs inoculated.

Bastai,[10] working in the clinic of Micheli, found a filtrable virus in the central nervous system, obtained at necropsy, from cases of epidemic encephalitis. Subdural injection of this caused a fatal illness in rabbits, dogs, cats, guinea-pigs and rats, which was very similar to the disease in man. Characteristic microscopic cerebral lesions were present in the animals. The disease could be communicated indefinitely from animal to animal. With tissue-ascitic fluid medium, an extremely minute,

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7. Maggiore, S., and Sindoni, M.: Etiology of Epidemic Encephalitis, Pediatria, Naples **28**:985, 1920. Epidemic Encephalitis, ibid. **29**:682, 1921.

8. Flexner, Simon, and Noguchi, Hideyo: Experiments on the Cultivation of the Micro-Organisms Causing Epidemic Poliomyelitis, Tr. Assn. Am. Physicians, Philadelphia **28**:55-68, 1913.

9. Ottolenghi, D.; d'Antona, S., and Tonietti, F.: Etiology of Lethargic Encephalitis, Policlinico, Rome **27**:1075, 1920.

10. Bastai, P.: Ricerche batteriologiche sperimentali intorno alla etiologia della encefalite epidemica, Sperimentale **76**:1, 1920.

filtrable organism was cultivated from Berkefeld filtrates of the original brain material obtained from cases of epidemic encephalitis and from the brains of the animals which succumbed to the experimental disease. Cultures of this organism, injected subdurally, produced in animals the same disease and the characteristic pathologic lesions already mentioned. Micheli,[11] says that he is convinced of the accuracy and correctness of the investigation of Bastai, which was performed in his clinic.

Kling, Davide and Liljenquist [12] recently reported confirmation of their previous findings [13] that the virus of epidemic encephalitis appears

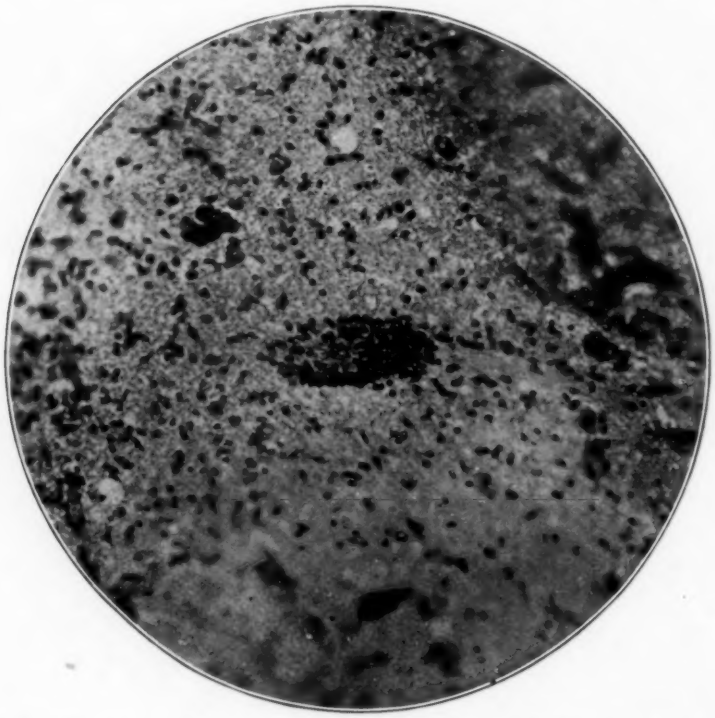

Fig. 1.—Midbrain region of a case of epidemic encephalitis, myoclonic type.

to be glycerol resistant, filtrable and uncultivatable. Their recent report was based on the inoculation of rabbits with spinal fluid from a case of epidemic encephalitis. Four rabbits were originally inoculated and were killed in from thirty-eight to forty days. The brains of two showed characteristic lesions. Five rabbits were inoculated with brain material

11. Micheli, F.: Intorno all'etiologia dell'encefalite Epidemica, Riforma méd. **37**:9, 1921.

12. Kling, C.; Davide, H., and Liljenquist, F.: Virus of Epidemic Encephalitis in the Cerebrospinal Fluid, Hygiea, Stockholm **83**:566 (Sept. 16) 1921.

13. Kling, C., and Liljenquist, F.: Compt. rend Soc. de biol. **84**:521, 1921.

from these. Two animals of this series were killed and the brains also showed characteristic lesions. They emphasize the possibility of employing inoculation of spinal fluid into rabbits as a diagnostic method. Netter, Cesari and Durand [14] have reported communication of the disease to rabbits by inoculation of nervous tissue from a man who died fifteen months after the onset of the disease. They were able to transfer the virus from animal to animal by inoculation of extract of the salivary glands. They found that the virus remained virulent in 50 per cent. of glycerol for at least thirty-two days.

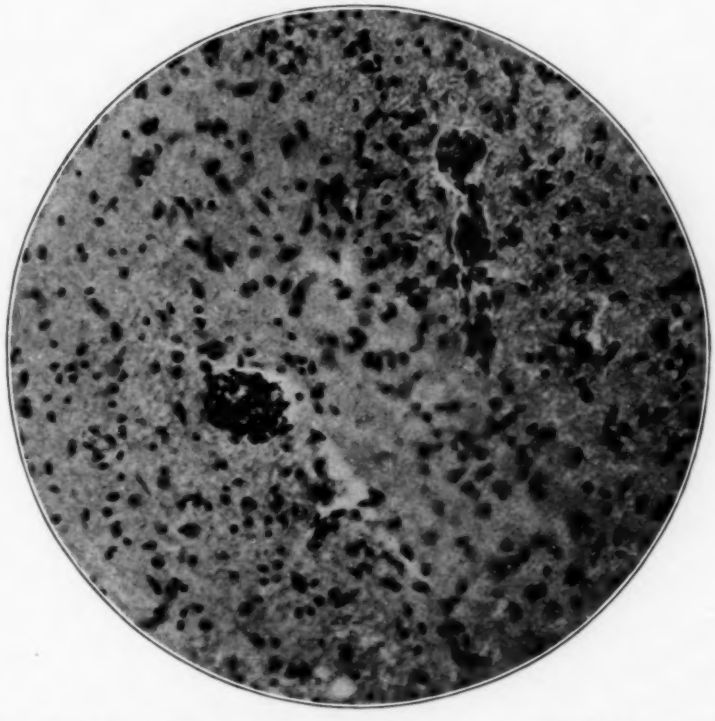

Fig. 2.—Midbrain region of a case of epidemic encephalitis, myoclonic type.

Amoss [15] found that serum from convalescing cases of epidemic encephalitis did not protect monkeys from poliomyelitis virus, whereas serum from convalescent poliomyelitis cases does protect monkeys from this virus. This immunologic difference is contrary to Maggiore and Sindoni's belief in the identity of poliomyelitis and epidemic encephalitis

14. Netter, A.; Cesari, E., and Durand, H.: Demonstration de l'activité du virus dans les centres nerveux 15 mois apres le debut. Presence de ce virus daus les glandes salivaires, Compt. rend Soc. de biol. **84**:854, 1921.

15. Amoss, H. L.: Immunological Distinctions of Encephalitis and Poliomyelitis, J. Exper. Med. **33**:187, 1921.

and is further verification that these two diseases are separate entities caused by viruses which biologically are closely related.

Several other communications are of interest, although their exact significance cannot be determined. Doerr and Schnabel,[16] and Blanc and Caminopetros[17] found a filtrable virus in the plasma of herpes febrilis which caused herpetic keratitis when injected into the cornea of rabbits. This condition can be transferred from animal to animal. This virus, when injected subdurally

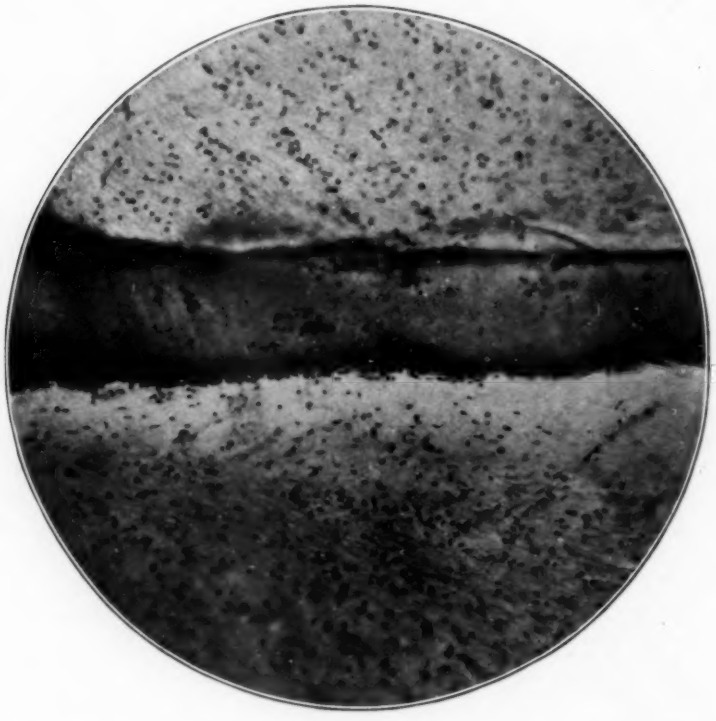

Fig. 3.—Subcortical region of a case of epidemic encephalitis, fulminating type.

into rabbits, is said to produce clinical and pathologic manifestations identical with those produced by virus from cases of epidemic encephalitis. They believe these two viruses are closely related. Luger and Lauda[18] report finding

16. Doerr, R., and Schnabel, A.: Das Virus des Herpes febrilis and seine Beziehungen zum Virus der Encephalitis epidemica (lethargica), Schweiz. med. Wchnschr. **51**:469 (May 19) 1921. Weitere experimentelle Beitrage zur Aetiologie und Verbreitungsart des Herpes febrilis beim Menschen, ibid. p. 562.

17. Blanc, G., and Caminopetros, J.: Recherches experimentales sur l'herpes, Compt. rend. Soc. de biol. **84**:629-630, 767-770, 859-860, 1921.

18. Luger, A., and Lauda, E.: Zur Aetiologie des Herpes Febrilis, Ztschr. f. d. ges. exper. Med. **24**:19, 1921.

a filtrable virus in the fluid of the vesicles of herpes febrilis which on injection produced herpes in the cornea of animals. This condition can be transmitted indefinitely from animal to animal.

MATERIAL AND METHODS

The experimental and cultural methods used throughout this investigation were the same as those described in the preliminary communication[1] and identical with those previously described by Loewe and Strauss.[2]

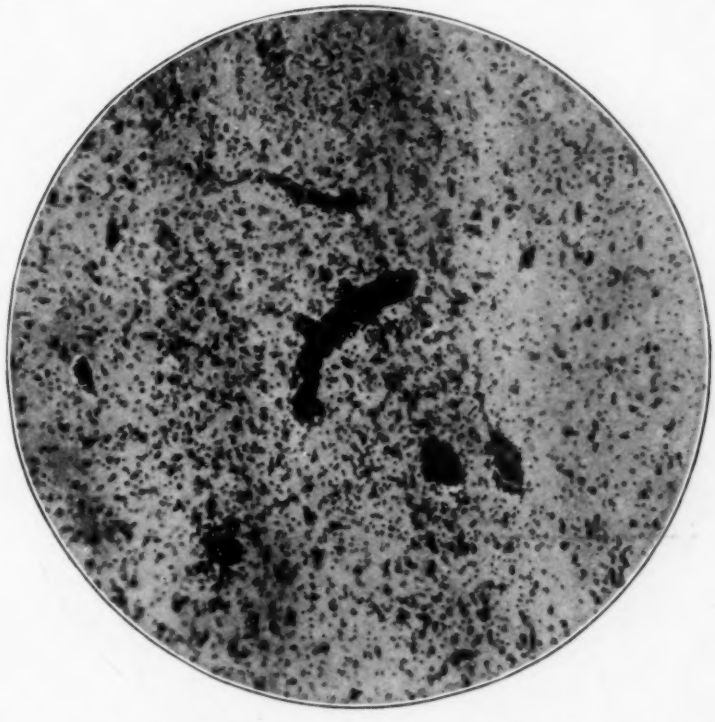

Fig. 4.—Brain of a rabbit inoculated intracranially with virus which had been passed through another rabbit.

The material and experiments are collected in the table. Animal inoculations referred to as positive are those in which microscopic brain lesions were found, identical with those present in fatal human cases. The inoculations were made intracranially. Positive cultures indicate recovery in the tissue-ascites medium, perfected by Noguchi, of a minute filtrable organism having the same morphologic, cultural and biologic characteristics as those recovered by Loewe and Strauss.[2]

The cases which came to necropsy ran typical clinical courses and varied from a fulminating case, with death eighteen hours after the first symptoms, to one of lethargic type which lasted eighteen months with remissions and relapses.

The brains in all these cases showed characteristic lesions; that is, perivascular round cell infiltration and focalized areas of degeneration and round cell infiltration, most numerous in the pons and midbrain region. In the two fulminating cases the lesions were definite but few in number. This is of importance since, in the cases studied by McIntosh and Turnbull, no lesions were found. Some of the brains from rabbits, which in the present study succumbed after inoculation, showed no lesions even though the virus was present, as was proved by culture and by transmission to other rabbits.

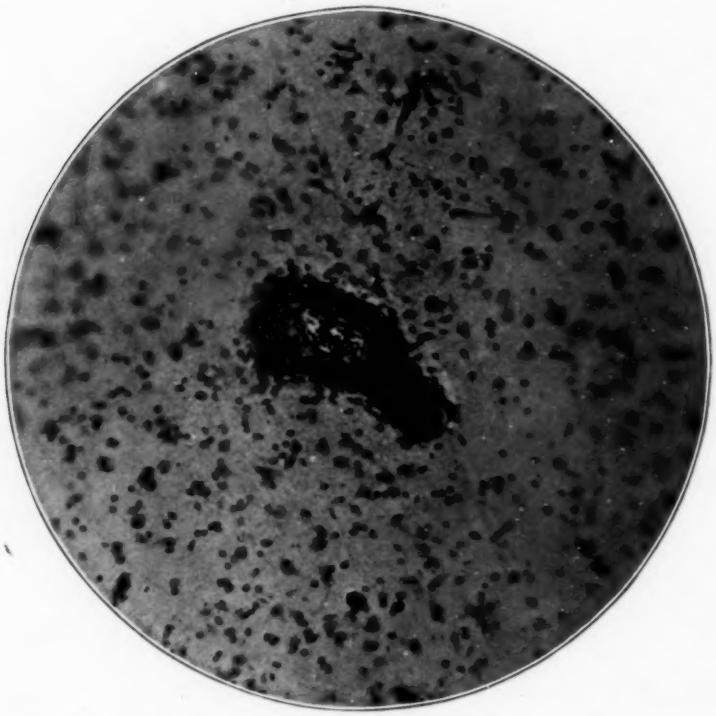

Fig. 5.—Brain of a rabbit inoculated intracranially with virus which had been passed through three other rabbits.

Necropsies were performed on seven cases of epidemic encephalitis. Filtrates (through Mandler clay filters tested to hold back *B. prodigiosus*) from five brains yielded positive animal inoculations and cultures. One brain filtrate yielded only positive cultures and one brain only positive animal inoculation, but from these animals the minute filtrable organism was recovered. Positive results, including both cultures and animal inoculation, were obtained in 100 per cent. of the seven brains studied.

The number of spinal fluids investigated was forty-five, secured from thirty-five cases. Positive cultures were obtained from spinal fluid from twenty-four cases, which is 70 per cent. of the cases and 53 per cent. of the spinal fluids examined. Positive animal inoculation was secured with spinal fluid from twenty-two cases, which is 65 per cent. of the cases and 49 per cent. of the spinal fluids studied. With spinal fluids from five (15 per cent.) of the cases animal inoculations were positive, although cultures were negative. Positive cultures were

Fig. 6.—Rabbit inoculated intracranially with culture of the minute organism in its third generation.

obtained from spinal fluids obtained on two different days from three cases. In two other instances the first spinal fluid, obtained when the patient was acutely ill, yielded positive cultures while subsequently, during convalescence, negative results were obtained from specimens of spinal fluid. In two cases, spinal fluid obtained three and five months after onset of this disease yielded positive cultures. The total of positive results, by both cultures and animal inoculations, was obtained with spinal fluids from 85 per cent. of the cases studied.

Nasopharyngeal washings were similarly tested from five cases; positive animal inoculation was obtained in all five, positive cultures in four. The combined results were positive in 100 per cent. in this series.

The total number of rabbits inoculated intracranially with either original material or filtrates of the brain of animals which succumbed was 295. Of these 167 died; 108, or 65 per cent., showed characteristic

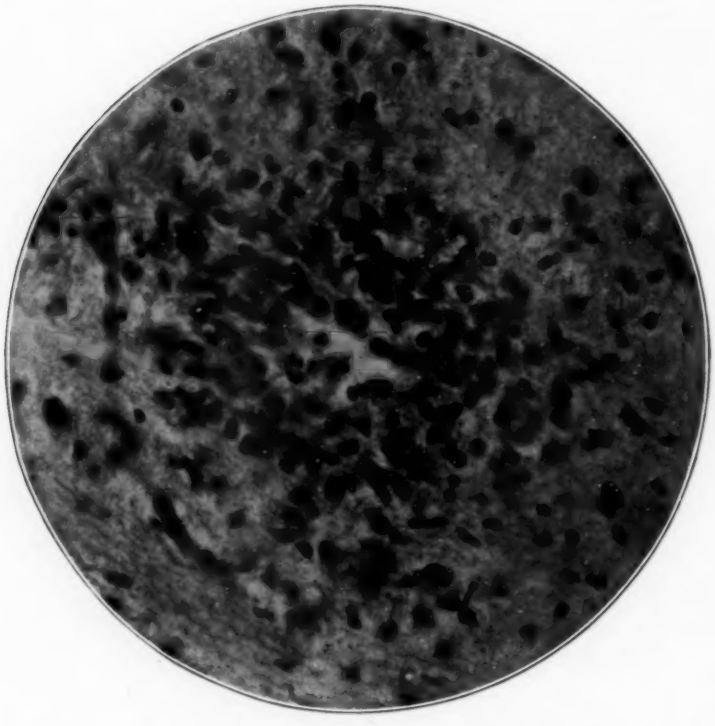

Fig. 7.—Rabbit inoculated intracranially with culture of the minute organism in its third generation.

lesions of epidemic encephalitis (only two rabbits died from acute meningitis). Most of the strains of virus were passed through only one or two series of animals, but passage of a few strains was made through twelve, five, five and four series.

The animals generally died in from two to eight weeks after inoculation, and this is considered to be the incubation period after intracranial inoculation. A few rabbits died in from one to two days. Death after this short interval was believed to be caused by some factor other than the development of the disease, as cerebral lesions were not present. Nevertheless, in a number of instances when the

animal, inoculated intracranially with a potent virus, succumbed after a day, cultures of brain filtrate and transmission experiments were positive.

Viruses of different origins were inoculated intracranially into fifty-two guinea-pigs; thirty-five died, and twenty-one, or 60 per cent., showed characteristic brain lesions (three guinea-pigs died from acute meningitis).

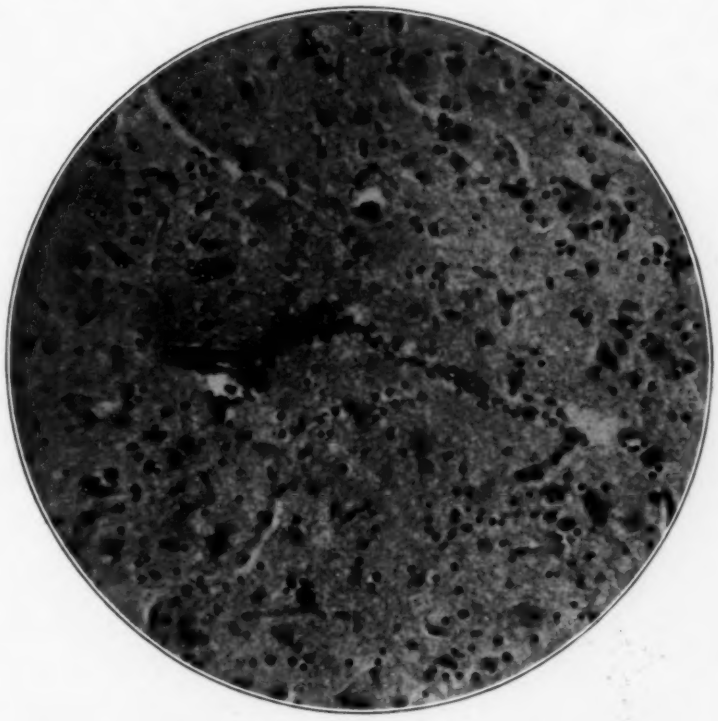

Fig. 8.—Rabbit inoculated intracranially with culture of the minute organism in its third generation.

Cultures in the third, fifth and fifteenth generations were inoculated intracranially into rabbits with the same percentage of positive results as with injections of virus, and the minute organism was again recovered from the brain filtrate of the animals that died.

Some of the cultures were carried through twenty-two generations. Cultures were repeatedly filtered and the minute organism recovered from the filtrate.

Spinal fluid from the following control cases and brain filtrates from all but the last two were inoculated intracranially into thirty rabbits with negative results: three cases of glioma of brain, one case each of

tuberculous meningitis (verified at necropsy), Hodgkin's disease, poliomyelitis, staphylococcus bacteremia, tubercle of cord (verified microscopically) and diabetes.

Cultures on ordinary mediums of these spinal fluids and brain filtrates yielded no growth. Most of the spinal fluids, brain filtrates and filtered nasopharyngeal washings were similarly cultivated and showed no growth, except an occasional spinal fluid from which an obvious contamination was recovered. Filtrates from the rabbit brains yielded no growth.

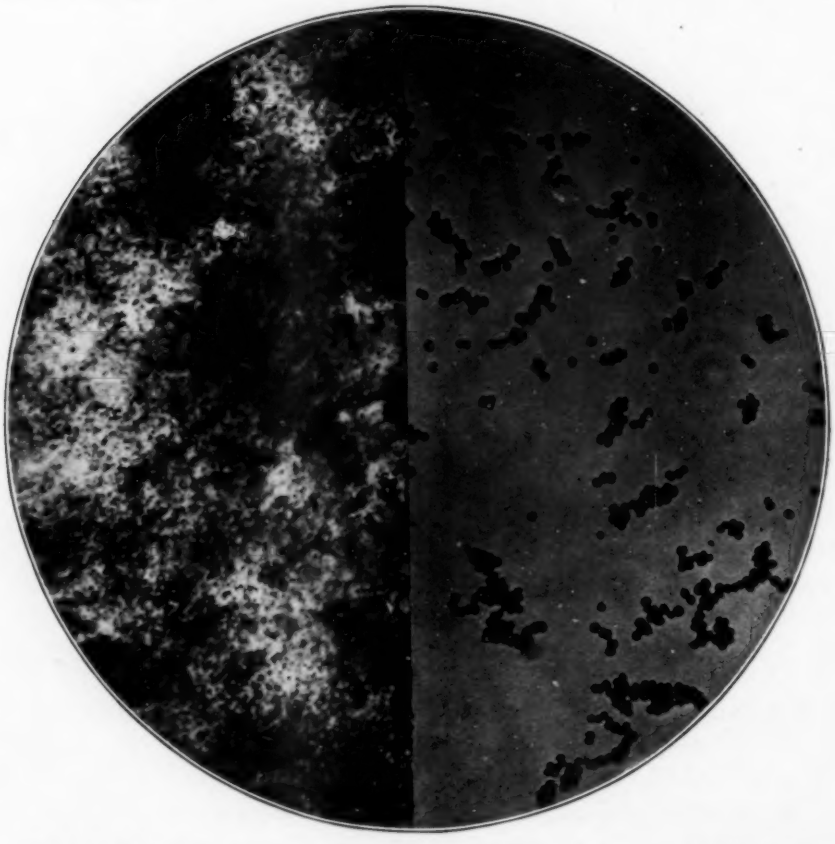

Fig. 9.—This illustration shows the comparative size of *Staphylococcus aureus* and the minute filtrable organism cultivated in Noguchi medium from the brain filtrate of a case of epidemic encephalitis; × 1200.

Although the human brains were not removed aseptically at necropsy, nevertheless two brains were cultivated on ordinary mediums (including blood agar) and in deep tubes (which had been autoclaved) of glucose bouillon containing a piece of sheep brain. The surface of the brain was seared and pieces were removed aseptically from the cere-

brum and midbrain. Seven rabbit brains, removed aseptically, were similarly cultivated. Most of the tubes inoculated with the human brains showed growths of saprophytic bacteria. Only an occasional tube inoculated with rabbit brain yielded growth, usually *Staphylococcus aureus*. Streptococci (hemolytic or anhemolytic) were never recovered from these blocks of brain, brain filtrates (human and animal), spinal fluids or filtered nasopharyngeal washings. Sections from the brains of five cases and from numerous rabbit brains showing characteristic lesions were stained by the Gram-Weigert method and no cocci or other organisms were found.

RESULTS OF EXPERIMENTS

	Number of Cases	Number of Specimens	Number of Positive Animal Inoculations	Number of Positive Cultures	Negative Cultures but Positive Animal Inoculations	Total Number of Results, Both Cultures and Animal Inoculations
Brain filtrate (necropsy material)	7	7	6 (Case 5 was negative)	6 (Case 6 was negative)	..	100%
Spinal fluid	34 Including 7 mild cases and 8 convalescing cases	45	22 65% of cases 49% of spinal fluids	24 70% of cases 53% of spinal fluids	5 15% of cases	85%
Nasopharyngeal washings	..	5	5 100% of specimens	4 80% of specimens	1 20% of specimens	100%

Control, uninoculated rabbits were sacrificed from time to time from different groups of animals. The animals were secured in lots of one or two dozen from different stocks and dealers. None of the brains from these animals showed gross or microscopic lesions. Some of the inoculated animals which died after several weeks, or remained alive, developed snuffles. Two animals which developed snuffles were sacrificed and the brains found normal, grossly and microscopically. Of those which died after inoculation and which had developed snuffles some showed lesions characteristic of epidemic encephalitis and some did not. It is, therefore, not believed that snuffles in rabbits can account for the microscopic brain lesions characteristic of epidemic encephalitis found in the animals used in this investigation.

SUMMARY

The investigations of epidemic encephalitis by Loewe and Strauss, which antedate other similar studies, seem to indicate that a filtrable, living agent, or virus, is regularly associated with this disease. This

virus causes a disease in animals which is similar to, and in many animals identical with, epidemic encephalitis. The microscopic cerebral lesions in the animals are the same as those which have been found to be characteristic of this disease. From this virus, an extremely minute filtrable organism was grown in the ascites-tissue culture medium perfected by Noguchi. Cultures of this organism likewise produced the characteristic disease and lesions in animals.

The study reported confirms the findings of Loewe and Strauss [2] in their entirety. Their animal experiments with the filtrable virus have been confirmed also by McIntosh and Turnbull,[4] Levaditi and Harvier,[6] Maggiore and Sindoni,[7] Ottolenghi, d' Antona and Tonietti,[9] Bastai,[10] Kling, Davide and Liljenquist,[12,13] and by Netter, Cesari and Durand; [14] and their cultural experiments, by Maggiore and Sindoni [7] and by Bastai.[10]

Abstracts from Current Literature

A GENERAL CONSIDERATION OF ENCEPHALITIS (MORPHOLOGY AND PATHOGENESIS). C. von Monakow, Schweizer Arch. f. Neurol. u. Psychiat. **10**:3, 1922.

The present article serves not so much to review the subject of encephalitis from an historical standpoint or to summarize the recent histopathologic work, but to express, in a more general way, the point of view gained by thirty years of study of encephalitis from the morphogenic and pathophysiologic standpoints. Among the causes of encephalitis may be enumerated trauma, compression, toxic factors, particularly the encephalitis of erosion, and most frequently infection.

Both the pathologic and the clinical picture may be exceedingly varied; the lesion may be focal or diffuse, with or without involvement of the meninges, suppurative or nonsuppurative, at times localized to the cortex, at times to the ventricle or the choroid plexus, sometimes parenchymatous, at other times mesodermal and with indirect injury to the nervous structures.

The present article deals with the infectious, nonsuppurative type. Among the causes are various types of cocci, bacilli, spirochetes, spirilli, ultrafiltrable organisms, etc. In addition to the acute hemorrhagic poliencephalitis of Wernicke, the encephalitis of syphilis and diphtheria, all of which have been known for a longer period of time, there have been added the encephalitis associated with Heine-Medin's disease, influenza, scarlet fever, chorea, rabies, chlorosis, herpes, and a variety of other conditions. Progressive paralysis and multiple sclerosis are essentially encephalitic in nature. Of outstanding interest at the present time is epidemic encephalitis. The difficulty of distinguishing toxic from infectious types is shown in the newly described guanidin encephalitis of Fuchs and Pollack.

The manner of invasion of these types, whether through the nasopharynx, the tonsils, the upper respiratory tract, the intestinal canal, the lymph or the blood stream has not yet been fully clarified.

The brain, probably more than any other organ, is protected by numerous structures and mechanisms. Nonsuppurative encephalitis represents that type of morphologic reaction in the brain in which the inflammatory process takes place almost entirely within the ectomesodermal barrier. Two great types can be distinguished: The first is essentially parenchymatous. It is characterized by a rather diffuse involvement, notably of the floor of the ventricles and of the brain stem, the cortex remaining relatively intact. If the latter is involved, it is not in the domain of special arteries, but takes place in an irregular manner, particularly in the gray substance. Microscopically, it is characterized by scattered, fairly sharply circumscribed areas of liquefaction. There is edema of the entire nerve structure, especially in the oblongata and the pons, venous stasis, a relatively uninvolved pia, slight changes in consistency of the brain substance, and here and there punctate extravasations of blood. The ventricles are only moderately filled with a clear fluid, although the choroid plexuses are swollen and hyperemic. The vena magna galleni and the veins of the stem are engorged. To this general type belong some cases of influenzal encephalitis, the encephalitis of chorea, rabies, typhus, scarlet

fever, herpes and epidemic encephalitis. Whether or not the epidemic variety is to be identified with the encephalitis of influenza cannot be determined, although Economo insists that they are etiologically quite different.

Of this infectious, nonsuppurative, parenchymatous type one can distinguish two subdivisions. In one, the brain substance directly suffers a diffuse involvement; in the other, involvement is indirect and through the blood vessels. The manifest pathologic process takes place chiefly in the floors of the ventricles, the gray matter about the aqueduct of Sylvius, the thalamus and the striate regions; in other words, in the district most intimately related to the choroid plexus. The microscopic picture includes exudation, infiltration of vessel walls with lymphoid elements, abbau products of nerve cells and fibers, red blood cells, in the acute cases, neuronophagia, rosette formation, atrophy, degeneration, various stages of necrosis of nerve cells, distention of His' space, rarefication of structures under the pia. These findings are also noted in progressive paralysis, multiple sclerosis, rabies, chorea, herpes and a variety of other types of encephalitis.

Von Monakow then reviews briefly his theory of the nutritive functions of cerebrospinal fluid. The transportation of metabolic waste products, he thinks, takes place along two channels; one in which the cerebrospinal fluid carries off dissolved substances through the space of His, and from there to the subarachnoid space, the other in which insoluble products are carried through the agency of cells, through the perivascular space of Virchow-Robin, and thence in part through lymph spaces of the pia into the venous system, in part through the lymphatic glands of the head and neck.

Focal encephalitis, "Herdencephalitis," constitutes the second form and may be seen typically in influenzal encephalitis. In this type there is a more extensive involvement of the blood vessels and the formation of fairly well defined areas of softening and hemorrhages, variously located, but especially to be found in the white matter of the brain, the internal capsule, the pons, and the cerebellum. As opposed to the epidemic variety, the localization is in poorly vascularized areas. This type is found in poliomyelitis, in the encephalitis complicating ulcerative endocarditis, diphtheria, scarlet fever, syphilis, hemorrhagic encephalitis of Wernicke, and one type of influenzal encephalitis. According to Monakow, the arteriothrombotic basis of this form is well established. This does, however, not exclude an associated, diffuse parenchymatous degeneration of brain substance through the agency of toxins. The formation of thrombi explains the intermittent progression which is here so characteristic a feature. These thrombi are grayish white, attached to eroded endothelial surfaces, and characterized by a coraloid structure. As a rule, the lumen becomes entirely obliterated at this point, although the thrombus may be absorbed or give rise to emboli. Usually several days are required for its formation. The brain tissue thus deprived of blood behaves as in arteriosclerosis. It is possible that this thrombus formation is an expression of a defense mechanism on the part of nervous parenchyma against toxins circulating in the blood, and represents an effort to eliminate these from the supplied tissues. To be sure, this protection by means of thrombi can take place only at the expense of nerve tissue supplied by the vessel in question. This pathologic process was familiar to Monakow for twenty years, but was not, however, described previously. An accompanying photograph illustrates this point very well. The section is from the occipital lobe, evidently stained by the Weigert method. At one place is seen an old scar in the region supplied by the posterior cerebral artery. This is evidently the oldest lesion. At another place is an area of softening which

is fairly well advanced, evidently later in appearance than the one previously described. At a third place in the section is an area of early softening, of three days' duration. Recent thrombi are also shown in branches of the sylvian artery.

Clinically two varieties can be distinguished. In the first the course is strikingly intermittent and is characterized by apoplectiform seizures; this variety corresponds to the section just described. The second form is usually ushered in by a light apoplectiform attack and progresses continuously for a period of one or two years. The progression may be evidenced by vertigenous attacks, periodic disorientation, slowly progressive aphasic, agnostic or apractic symptoms. It may resemble progressive paralysis, however, with the distinction that in the latter the focal lesions are more pronounced, and the typical psychic picture is missing.

In encephalitis we deal with every combination of such processes. The more mechanical and focal lesions, caused by interruption of the continuity of nerve structures, expresses itself largely through reflex and coordination disturbances, localized cramps and convulsive movements, coarse choreic and athetoid phenomena and relatively insignificant disorientation in space and time after the initial stage. Disorientation in regard to personality, as a rule, is not an expression of this more grossly focal or mechanical type of lesion. Quite different is the clinical expression of a diffuse, toxic reaction. Here one finds delirium, soporous conditions, pains and abnormal sensations in the extremities, hallucinations, personal, spatial, and temporal disorientation, manic and depressive changes and apathy; in short, exceedingly varied disturbances in the psychic field. These split-off fragments many times do not fit, and compensation and adjustments are unusual.

Summarizing, Monakow would classify the nonsuppurative, infectious types as follows:

1. Diffuse, active, but eventually chronic parenchymatous type. In this type there are rarely missing focal lesions such as evidenced by ocular palsies; prominent in the symptomatology are soporose states, temporary periods of disorientation, delirium with hallucinations, depressive, manic, and schizophrenic episodes, pains, general weakness and a febrile course. These symptoms are particularly prominent in the initial stage; they may disappear without leaving a trace.

2. Focal Encephalitis: In this condition we are dealing largely with involvement of the mesodermal structures, that is, thrombosis, embolism and granulation limited to definite vascular territories, located particularly in the cerebrum. The brain parenchyma is involved secondarily. The expressions of disease are of more grossly localized character. This form is dominated, at the conclusion of the initial symptoms, by hemiplegic, paralytic or irritation phenomena. It is the type seen typically in influenzal encephalitis and a part of the ordinary cerebral palsy of childhood.

3. A type characterized by multiple, disseminated, usually well demarcated lesions of generally unknown, but probably infectious etiology. An example of this type is multiple sclerosis. The course is usually protracted and subject to marked fluctuations.

4. A chronic progressive type with diffuse involvement. This is almost certainly infectious in origin and includes progressive paralysis and the spirochetal types of encephalitis, such as trypanosomiasis.

WOLTMAN, Rochester, Minn.

PICTURES OF PARALYSIS AGITANS AND TETANY WITHIN THE SCOPE OF ARTERIOSCLEROSIS. P. MARTINI and A. ISSERLIN, Klin. Wchnschr. **1**:510 (March 11) 1922.

The authors report an interesting case of arteriosclerosis in which the signs of a paralysis agitans and of tetany occurred.

The patient, a female, was brought to the hospital, March 11, 1920. For four years prior to admission she had had periods of mental confusion. On examination she was found to be emaciated. The blood pressure was: systolic, 180; diastolic, 110. The face was masklike; there was stiffness of the lower limbs; and a rhythmic tremor of the head and upper limbs was noted both at rest and when in movement. The head was held forward, the pupils were small, and the nerve trunks tender. There was no Babinski sign. Mentally she was disoriented for time and place. In taking the blood pressure, it was noted that a typical Trousseau phenomena developed.

The dementia gradually progressed. The Trousseau sign persisted, but no other evidence of a latent tetany was found. On March 29, epileptiform attacks occurred, and on the following day aphasia developed. During the following two months the dementia lessened, the Trousseau phenomena disappeared, and the parkinsonian attitude became more marked. Death occurred on June 22, 1920.

At postmortem a marked peripheral and central arteriosclerosis was found. The blood vessels of the convexities and especially of the base of the brain showed severe sclerotic changes. In the region of the right praecuneus there was a recent hemorrhagic softening. The lateral ventricle was somewhat dilated. In the left lenticular nucleus there was a fissured softening extending into the internal capsule. The cerebellum and the temporal regions were essentially negative.

The interest in the case lay in the association of the three syndromes—cerebral arteriosclerosis, paralysis agitans and tetany, with the possibility of a similar underlying pathologic condition. Wilson, Lewy, and Forster have found lenticular lesions in paralysis agitans. MacCallum, on the other hand, considers the subcortical centers as a possible site of the pathologic condition in tetany. In the present case the parathyroid glands unfortunately were not studied microscopically, but grossly no changes were noted. The authors do not wish to place too much weight on the findings noted in this case as a possible solution of the problem.

The presence of paralysis agitans, an amyostatic disease, in conjunction with the tetany, brings the possibility of their relation closer together. The gait of the dog or cat in which the parathyroids have been removed is not unlike that obtained by Sherrington in cases of decerebrated rigidity. E. Frank has called attention to the vegetative nervous system in cases of amyostatic disturbances, and to vegetative disturbances in tetany. The authors feel that although nothing has been proved by this patient, attention should at least be called to the relationship between the diseases.

MOERSCH, Rochester, Minn.

MICROPTIC HALLUCINATIONS. RAOUL LEROY, l'Encephale **16**:504 (Nov.) 1921.

In general literature diminutive human beings are frequently mentioned. Perhaps the most famous description is in Swift's "Gulliver's Travels," in which Gulliver makes a voyage to Lilliput. In medical literature microptic

hallucinations (in the French "hallucinations lilliputiennes" and in the German "winzige Männchen") have been little mentioned. The author quotes from various sources descriptions of a peculiar sort of visual hallucination occurring in hypnogogic states, narcoses, cerebral arteriosclerosis, skull injuries, in toxic and febrile states, and associated with epilepsy as a psychic form of aura.

In these hallucinations the subject sees small human beings fantastically and brilliantly costumed and engaged in all sorts of activities. It is worthy of note that actual objects seen by the subject of these hallucinations are perceived as of normal size and color. Thus the state in which microptic hallucinations occur differs from the state of complete micropsia in which objective stimuli are all perceived on a diminutive scale. A characteristic of this type of hallucinations is that their emotional content is almost invariably agreeable.

Leroy regards these hallucinations as of importance in that they show clearly the relation between cortical centers and activities of the psyche. The former he regards as stimulated by a toxin which, at the same time, depresses consciousness. According to Theobald Smith, the reveries of children are occupied almost exclusively with play activities and fairy tales; many authors have described dreams of goblins and sprites and Lilliputian-sized landscapes. These things Leroy believes throw light on the source of microptic hallucinations. He regards them as produced by the subconscious or the unconscious, which is active, due to these toxins which at the same time depress normal consciousness but irritate cortical centers.

The pleasant emotional character of the hallucinations may be partly accounted for because the visions themselves are small and harmless, but particularly because the subject himself subconsciously believes in a world peopled by small beings who occupy themselves with agreeable pastimes. This principle explaining the agreeable emotional tone holds true also in the rare instances in which the hallucinations have as their content small demons and horrible activities which evoke terror because the subject has himself believed in such things.

The author does not mention the possibility of the peripheral visual apparatus participating in the production of these hallucinations.

Hyslop, New York.

SPINA BIFIDA OCCULTA AND SCIATICA. Hermann Weskott, Klin. Wchnschr. **1**:625 (March 25) 1922.

During the past few years much attention has been devoted to spina bifida Under the head of myelodysplasia, Fuchs correlated spina bifida occulta with such conditions as enuresis, club foot, prolapsed uterus and other malformations. Recently the association of sciatica with spina bifida has been considered by several authors. Gudzent has noted in numerous cases of sciatica, spina bifida occulta of the last lumbar or first sacral vertebra, and he is of the opinion that in older people the presence of a vertebral deformity may play some part in the causation of sciatica.

Peritz has tried to show that this type of sciatica can be differentiated from others by the presence of signs of myelodysplasia, such as club foot, enuresis, numbness and lancinating pains. The author states that in a series of 260 cases of sciatica, six cases showed spina bifida occulta. The cases all occurred in otherwise healthy men of middle age. The case reports are briefly included.

The histories in all six cases were essentially the same—insidious pain in the back and down one leg, gradually becoming more severe and extremely

chronic and resisting all treatment. In the author's series there was no history of early enuresis, there was no hypertrichosis, sensory disturbance or claw foot. Judging from his own experience, he is not at all certain that the presence of a spina bifida occulta is to be considered the cause of sciatica. It is, however, possible, that this acts as a locus minoris resistentiae. In some cases operative measures have been instituted, and deformities of the nerve roots have been noted; however the relationship between these findings and the sciatica is not clear. The majority of the author's cases responded poorly to treatment, and he suggests that in all chronic cases which are resistive to treatment the possibility of an occult spina bifida be considered. Whether operative procedure should be instituted in these chronic cases is still quite uncertain, and the writer does not feel that he is in a position to make any definite recommendations from his limited experience.

MOERSCH, Rochester, Minn.

SOME FACTS IN THE DEVELOPMENT OF THE AMPHIBIAN NERVOUS SYSTEM. C. JUDSON HERRICK, Anat. Rec. **23**:291, 1922.

The author discusses three factors which, among others, may be recognized in the embryologic development of the brain: (1) ancient palingenetic hereditary influences, such as primitive metamerism; (2) hereditary factors of more recent origin obviously of an adaptive nature, such as the reflex patterns and their neuromotor apparatus, and (3) the immediate effect of active function on the progress of individual development.

Until recently attention has been confined largely to the first of these factors, but the demand now is for a coordinated attack on the problems of development on all these frontiers. In order to learn the rôle of functional adaptations of the second and third types it is obviously first necessary to determine what these adaptations are. For the study of the development of functional localization and functional inter-relationships of parts, the amphibian nervous system is more suitable than the human brain, because it is less complex, more readily submits itself to the methods of experimental embryology and begins to function in response to external stimulation at a surprisingly early stage of development. A summary of the more recent work on the nervous system of amblystoma is given.

The author says that one of the first desiderata is a series of normal tables of the nervous system, based on the sequence of both structural and functional development. The object of the investigation is to correlate the changes in external form with corresponding changes in behavior and pattern and a histologic differentiation internally. For the consummation of this program more detailed studies of the development of reflex patterns from early larval stages are necessary.

NIXON, Minneapolis.

DISORDERS OF SENSATION IN PARALYSES OF THE FACIAL NERVE. J.-R. PIERRE, Presse méd. **30**:488 (June 7) 1922.

In certain cases of advanced cerebellopontile angle tumor, ageusia of the anterior two thirds of the tongue was demonstrated, opposing the classical theory that taste disorders were found only when the lesion lay between the geniculate ganglion and the origin of the chorda tympani.

In refrigeration palsies, diminution in the sense of taste varied directly with the degree of paralysis, but ageusia was the more persistent of the two symp-

toms. The anterior two thirds of the tongue frequently showed thermal hypesthesia in addition, which disappeared sooner than the gustatory hypesthesia.

Hypesthesia of the concha auriculae, earlier associated with the facial nerve in herpetic cases, was here found in several cases without herpes. This tended to clear up by the fourth to the sixth day. It is announced that Truffert has demonstrated fibers in an anatomic preparation which are identified as the terminal sensory branches of the seventh nerve to this area.

Classic painful hyperacusis at the onset of a refrigeration palsy was found in only 6 per cent. of the cases studied. Lachrymal, buccal and sweat secretions were rarely, and then only slightly, affected.

HUDDLESON, New York.

THE DIAGNOSIS OF HYSTERIA. HENRY HEAD, Brit. M. J. **1**:827 (May 27) 1922.

Head regards hysteria clinically as definitely characterized by the positive nature of its morbid phenomena. For instance, an hysterical paralysis is the expression of the conviction "I cannot move" in the mind of the patient. Likewise, other deficit symptoms and signs, such as anesthesia, result from the refusal to accept impressions. The basic mental factors are proneness to autosuggestion, a negative attitude to orders from without and a tendency to disassociation. Hysteria is essentially an irrational answer to a mental conflict. Head's therapeutic advice is well worth repetition. "It must never be forgotten that in a large number of cases, especially in civil life, removal of hysterical symptoms is only a prelude to the discovery of an anxiety neurosis. The causes for the suppressed emotion must be investigated, or the patient may be left in an even worse condition than that in which you found him."

STRECKER, Philadelphia.

THE RADICULAR DISTRIBUTION OF NEVI AND VITILIGO. M. KLIPPEL and M. P. WEIL, Presse méd. **30**:388 (May 6) 1922.

The two disorders are considered together, with arguments for their common origin. Several cuts illustrate the root distribution of areas of vitiligo.

Nevi are divided into (1) those due to a peripheral lesion, in the vessel walls, which may occur early embryologically, and (2) those due to central lesions, in the brain or cord, that can occur effectively only late in fetal life when the nervous system is in trophic connection with the integument.

Certain nevi and leukodermic patches are apparently due to lesions in the peripheral nerves, but it is claimed that the majority are dependent on central nervous lesions. The latter are located in the posterior commissure of the spinal cord. Vitiligo is said to be frequently accompanied by an excess of globulin and even of cells in the spinal fluid, though not necessarily syphilitic.

HUDDLESON, New York.

THE SEMEIOLOGY OF INVOLUNTARY RHYTHMIC MOVEMENTS IN EPIDEMIC ENCEPHALITIS. M. E. KREBS, Progrès méd., April, 1922, No. 13, p. 145.

This paper describes unusual and abnormal forms of movement in epidemic encephalitis. Krebs reports seven cases. He is particularly interested in scoliosis. He quotes a previous paper on this subject in the *Revue*

neurologique, March, 1922, written with Babinski and Plichet. Other movements of the extremities are reported. Many of these are fragments of decerebrate rigidity. He emphasizes the great effect of emotion in exaggerating the extent not only of the trunk abnormalities, but also of the movements of the extremities. Finally, emphasis is laid on the similarity of the movements in epidemic encephalitis to those of athetosis, chorea and those cases designated torsion spasm by Ziehen and dysbasia lordotica progressiva by Oppenheim.

KRAUS, New York.

THE INFLUENCE OF THE LATERAL LINE SYSTEM ON THE PERIPHERAL OSSEOUS ELEMENTS OF FISHES AND AMPHIBIA. ROY L. MOODIE, J. Comp. Neurol. **34**:319 (June 15) 1922.

The lateral line sense organs are not trophic for the head skeleton. The association of lateral line canals with certain definite peripheral osseous elements has been a constant feature in the organization of fishes and Amphibia from Devonian times to the present. The nature of the influence of the lateral line canals on the formation of bone is mechanical, because it furnishes an inactive substance in the form of the dense connective tissue of which the canals are composed. Attention is called to some analogous processes of calcification in human neuropathology.

C. J. HERRICK, Chicago.

FURTHER COMPARATIVE STUDIES IN OTHER FISHES OF CELLS THAT ARE HOMOLOGOUS TO THE LARGE IRREGULAR GLANDULAR CELLS IN THE SPINAL CORD OF THE SKATES. CARL CASKEY SPEIDEL, J. Comp. Neurol. **34**:303 (June 15) 1922.

Dahlgren has described, in the spinal cord of the skate, enormous cells of nervous origin, but apparently of glandular function. Examination shows that homologous cells are found in the spinal cords of most (but not all) groups of fishes. Many gradations are described between cells which in size and internal structure resemble ordinary neurons and the large and peculiar cells of skates and flounders.

C. J. HERRICK, Chicago.

CLINICAL CHARACTERISTICS OF MUSCULAR TWITCHINGS IN EPIDEMIC ENCEPHALITIS. THE RELATIONSHIP OF ACTUALLY OBSERVED MYOCLONIAS TO SOME PREVIOUSLY KNOWN SYNDROMES. RHYTHMIC SPASMODIC MOVEMENTS IN ENCEPHALITIS. M. E. KREBS, Bull. et mém. Soc. méd. d. hôp. de Paris. March 24, 1922.

Krebs emphasizes the difference between rhythmic and arrhythmic myoclonic movements. He says that both types exist in epidemic encephalitis. The arrhythmic type recalls the contractions of the paramyoclonus of Friedreich. The rhythmic type differs from the arrhythmic in that it moves the limbs. Scoliosis and various movements of the extremities are produced in the rhythmic type.

KRAUS, New York.

CYTOLOGY OF THE LARGE NERVE CELLS OF THE CRAYFISH (CAMBARUS). L. S. Ross, J. Comp. Neurol. **34**:37 (Feb. 15) 1922.

The neurofibrillae of the axons of these cells take a remarkable course within the cell body, spreading widely throughout the cytoplasm and enveloping the

nucleus. These fibrillae are not related to trophospongium of Holmgren, internal reticular apparatus of Golgi, or mitochondria.

C. J. HERRICK, Chicago.

THE MOTOR NUCLEI OF THE CEREBRAL NERVES IN PHYLOGENY. A STUDY OF THE PHENOMENA OF NEUROBIOTAXIS. DAVIDSON BLACK, J. Comp. Neurol. **34**:233 (April 15) 1922.

This is the fourth of a series of studies, of which the preceding parts have appeared at intervals during the past five years. The motor nuclear pattern of birds is on the whole fundamentally similar in all the forms examined and characteristically different from that of any other vertebrate group. The special features of the motor nuclei of several avian types are described in correlation with the structural peculiarities and modes of life of these species.

C. J. HERRICK, Chicago.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

Regular Meeting, April 4, 1922

FOSTER KENNEDY, M.D., *President, in the Chair*

PATHOLOGIC FINDINGS IN THE HEART IN PROGRESSIVE MUSCULAR DYSTROPHY. DR. JOSEPH H. GLOBUS.

This paper and discussion will appear in full in a future issue of this journal.

A NEUROPSYCHIATRIC PILGRIMAGE. Illustrated by lantern slides. DR. SMITH ELY JELLIFFE.

In May of 1921, I had the privilege of visiting many of my old European haunts, and it is of this journey I wish to speak.

France lost at least three of its greatest neurologists during the war. Its sister state, Belgium, also suffered an irreparable loss. They were all older men, and each had done his life work. Each stood preeminent in his sphere. Déjerine was perhaps the greatest figure of them all. I need not remind this audience what he means to neurologic science. His early "Famille Neuropathique," his "Familial Myopathy," are milestones in neurologic progress. With Thomas, his "Maladies de la Moelle" is a classic; his "Anatomie," written in conjunction with Mme. Déjerine, and his final large volume on "Semeiologie"—these are only a few of his standard performances which have enriched neurologic science. I would like to speak of his personal charm, his bonhomie, his rare skill and tact in handling neurotic patients according to his view of their disturbed emotional conditions.

Another lost leader, a native of Bordeaux, who has been one of the dominating figures in French psychiatry for many years is Régis. His name is familiar to many of the older men in psychiatry. He was a fearless and strong man. He was one of the first to be interested in psychanalysis, and with his pupil, now his successor, Hésnard, in Bordeaux, gave us their well-known criticism of the freudian principles.

In the early years of the war the death of Van Gehuchten made all who had known this gentle soul, mourn. His life work in Louvain had been destroyed in that mad rush of war and he himself could not survive it.

Our next immortal, Grasset, was a remarkable neurologist, and the younger men here may profit from Grasset and Rauzier's "Traité de Neurologie," a large book, crowded with data and quite the equal in many respects to Oppenheim's classic.

Having paid short respects to the dead, let us return to the primary object of my visit, namely, the annual reunion of the Paris Neurological Society—1920 had seen the first general reassemblage of that body since 1914.

This leads our steps to that great Mecca of French Neurology, the "Salpetrière." Here for many generations neurology and psychiatry have

drunk deep of knowledge, reaching an acme in the genius of Charcot. Raymond, his successor, carried on, feebly perhaps, in view of his predecessor's brilliancy, followed later by Déjerine. At his death in 1918, Pierre Marie followed him and it is owing to his courtesy that I am able to show you an intimate glimpse of the Charcot library and collections housed in the building in which the clinical work is carried on. Pierre Marie, the present professor of neurology, is remarkable for his facile and intelligent interest in all things neurologic, especially his quick vision to grasp the significance of small variations in structure and function. A striking trait is his great courtesy to American students; the enthusiasm that he can arouse in his students makes him at present the most dominant figure in French neurology.

The nomination of Claude to the chair of mental medicine of the University of Paris has met with approval by his colleagues and confrères. In the field of neurology he has gathered ample harvests. In psychiatry it cannot be said that Claude has made, as yet, any striking contribution, but the solid foundations on which he has reared his knowledge of the action of human beings leaves little doubt that in this field he has much to contribute.

Henri Claude was made intern of the hospitals during the year of 1893, intern of the gold medal in 1896, doctor of the hospitals in 1901, fellow (agrégé) in neurology in 1903, and assistant at the Clinic of Nervous Diseases; under this title he was frequently in charge of the course at the Salpetrière, where he directed the service for nonrèsident psychopathic patients.

Claude has frequently been laureate of the Faculty of Medicine, of the Academy of Medicine and of the Academy of Sciences; he is a member of the societies of biology, psychiatry and legal medicine, as well as neurology, of which he has been president. For the last fifteen years he has been expert of the tribunals where association with him is particularly appreciated by both judges and physicians.

He has published a number of studies in *L'Encephale*, of which he is a director. It would be unjust to forget the services which he rendered during the war as chief of important neuropsychiatric centers and a director of commissions. It is impossible to give an exact idea of Henri Claude's works in the limited space of this talk. His publications touch on the broader problems of medicine, neuropsychiatry, endocrinology and experimental pathology. His studies on the pluriglandular syndromes, on the method of glandular tests and on the relations of the glands of internal secretion to disorders of the nervous system are known to most present. His book on the semeiology of division of peripheral nerves, enriched with valuable personal documents, was very useful in the study of injuries of nerves in the war. Serous meningitis and the syndrome of intracranial hypertension constitutes one of the most important works in neurology. His researches in cerebral tumors, epidemic encephalitis atrophy of the cerebellum, tumors of the protuberance, spinal disorders, sections of the spinal cord and other conditions are also valuable.

In psychiatry, his reports at conferences on epilepsy, the nature of hysteria, the rôle of the emotions in the psychoneuroses, apraxia, mental disturbances in epidemic encephalitis, dementia praecox and senile dementia made him an authority. If the moment has come when psychiatry is able to comprehend more than subtle classifications; if it can perhaps be impregnated with ideas of internal pathology, general pathology, neurology and endocrinology; then we may hope that Henri Claude, aided by the disciples he has already made and by others who may attach themselves to his school, will contribute powerfully to the renovation of this science.

Taking up the work of the Congress itself, Dr. Jelliffe spoke briefly of the reports by Souques on paralysis agitans, the functions of the paleostriatum and neostriatum; the substantia nigra; encephalitis and its lessons; and the lack of real understanding of what is meant by emotional factors. Roussy's brilliant work on the thalamus and many others were rapidly alluded to.

He then sketched the personalities of, and work done by, Leri, Bouttier, Guillain, Crouzon, Sainton, Vurpas, Bourguignon, Behague, Sicard, Foix, Laignel Lavastine and other representatives of present day Paris neurology. Mme. Déjerine and Ceillier's work in the osteo-arthropathies and Thomas' work on the pilomotor reflexes were shown by lantern slides.

Dr. Jelliffe then took his auditors to Switzerland, stopping with Dr. Robert Bing at Basel; and then to von Monakow's collections in Zurich, giving illustrations of von Monakow's ideas of the integration of bodily function, the choroid plexus and its relations to mental and nervous disease. A rapid visit was made to Bürgholgli Hospital and to Prof. Bleuler, and the present tendencies of psychanalytic applications to psychiatry were touched on.

He next turned to the Neurological Institute of Vienna, and Marburg, Pollak and Spiegel. Particular attention was given to Spiegel's recent work on the vegetative nervous system, to the work of von Economo and of Wagner, von Jauregg and the malarial treatment of paresis. The technic and the patients whom he had seen were described. Allusions were also made to later discussions at Braunschweig with Weygandt, Nonne and others whose results were encouraging.

The speaker then took his hearers to Kraepelin's clinic at Munich. He spoke of the death of Alzheimer, Nissl and Brodmann and gave short accounts of these workers. Kraepelin himself was seen in Italy; his energies are now directed toward building up his Research Institute; in this work he has an able ally in Rudin. The scientific work in the clinic is as active as ever. Spielmeyer and Spatz are carrying on the Alzheimer traditions and enlarging the scope of their investigations beyond purely cellular alterations.

Berlin was rapidly visited, and a brief résumé given of the work of Rothmann, Lewandowsky, Oppenheim and Erb. A visit to the Vogts was described and the extension work of the Vogts and Bielschowsky outlined. Vogt's program for work to be done on the cortex was briefly discussed and the work on the striatum outlined.

Dr. Jelliffe then gave a rapid summary of the Braunschweig meeting of the Deutsche Nervenerzte. Strümpell's amyostatic syndrome was the subject, and comparisons were drawn between the work at Paris and Braunschweig on the physiopathology of the region of the striatum.

Dr. Jelliffe dwelt for a moment on the fascinating work of Lewy on experimental studies of metabolic pathways of vegetative function. Lewy, with Brugsch and Dresel have commenced a direct attack on the neurology of metabolism and have shown the importance of mesencephalic structures for the integration of visceral functioning.

Dr. Jelliffe then took his auditors to London and the Queen Square Hospital. He reviewed rapidly Head's aspects of the problem of aphasia and spoke of the Head-Riddoch work on the mass reflex, bringing the latter into coordination with the studies of Lhermitte on the cord, and of André Thomas on the pilomotor reflexes.

He spoke of the death of Henry Maudsley, almost the only representative of English psychiatry who spoke in the language of dynamic psychology, and of his contributions to psychiatry. Only with the great war did English

psychiatry awake from an inertia that was difficult to understand. Stoddard, among the older group, alone seemed to comprehend the real situations as Maudsley had seen them. Mercier's crabbed satire had seemed to cramp psychiatry in England almost as effectually as he himself had been cramped by his venom and his rigid "logic," both of which he used to ridicule his adversaries.

That English psychiatry had begun to awaken was evident and was illustrated by Sir Frederick Mott's work on the gonadal changes in dementia praecox. Almost every endocrine organ has been indicted by different investigators. Undoubtedly the most radical alterations were to be expected in spermatogenesis and ovogenesis, and even in the cells of Leydig—all of which Mott found gravely altered. Whereas Mott argued chiefly for these gonadal changes as of primary significance, Dr. Jelliffe emphasized his belief that they were results and not causes. When, to use Southard's phrase, a fourth dimensional psychiatry becomes thinkable, the life movement of the organism as a whole must occupy the focus of attention. This dynamic urge, like time, forces the individual along lines of behavior, metabolic or social, and has a definite entelechy. The continuance of life is life's chief function. This has been written into every cell of the body and is of the essence of its expression. Naturally the gonadal system, more perhaps than any other structures, must record this push.

Nature's great aim may be conceived to be the development of adult psychosexual persons. Practically all mankind is struggling along this pathway and halting at various levels of psychosexual evolution. The chief criteria to determine the stage of this development in any individual case are found in the unconscious. The psychanalytic technic alone can determine this. All previously orthodox criteria of so-called group logic are usually camouflage, substitute products. In the study of unconscious processes one may be able, in a manner analogous to that used by the paleontologist, to determine a geologic horizon, to discover just what stage the individual has reached in his psychosexual evolution. His dynamic strivings bear a direct relationship to this grade of development, and his constitutional diseases, speaking in general, develop in definite associations with his dynamic strivings.

Dr. Jelliffe then discussed the work of S. A. K. Wilson and the striatum syndromes, bringing this author's contributions in line with the Paris Neurological Conference to which Wilson himself had contributed, and to the Braunschweig meeting where the same subject was discussed.

Three Hundred and Ninety-Seventh Regular Meeting, June 6, 1922

E. G. Zabriskie, M.D., *Vice President, in the Chair*

THE STATESTHETIC AND KINESTHETIC COMPONENTS OF THE AFFERENT SYSTEM. Dr. J. Ramsay Hunt.

The whole efferent nervous system, cerebrospinal and vegetative, consists of two components which are physiologically and anatomically distinct. One is the static system which regulates posture; the other is the kinetic system controlling movement itself. In muscle, the kinetic system innervates the

anisotropic contractile mechanism and the static system the isotropic sarcoplasm. The function of the sarcostyles is movement. The function of sarcoplasm is postural fixation, posture following movement like a shadow.

The efferent system, phylogenetically considered, consists of three great physiological divisions, which I term archeokinetic, paleokinetic and neokinetic. The segmental nervous system contains the archeokinetic and archeostatic components of motility, representing reflex movement and reflex posture (archeokinesis).

The paleokinetic and neokinetic mechanisms for the regulation of higher types of movement are found in the suprasegmental nervous system. The paleokinetic mechanism consists of the corpus striatum, its subordinate spinal systems (extrapyramidal tracts), and cortical connections through the optic thalamus (corticopaleokinetic system). This system controls movements of the automatic-associated type (paleokinesis). The neokinetic mechanism consists of the rolandic area and its corticospinal system (pyramidal tracts). The neokinetic system is concerned with isolated-synergic types of movement (neokinesis).

I regard the cerebellum as the essential suprasegmental structure for the regulation of postural function. Both neostatic (isolated-synergic) and paleostatic (automatic-associated) types of posture are represented. The neostatic function is related to the hemispheric system and the paleostatic to the vermian system of the cerebellum.

In peripheral nerves and voluntary muscle the myokinetic system is represented by medullated nerves and the sarcostyles of the muscle fiber, the myostatic system by nonmedullated nerve fibers and sarcoplasm. Each system has its special form of tonus, a contractile tonus, referable to the kinetic mechanism (kinetotonus) and a plastic tonus referable to the static system (statotonus).

The static and kinetic components of the vegetative nervous system for the control of unstriped muscle are represented in the sympathetic and parasympathetic systems respectively. These two systems control the postural function and primitive motility of the blood vessels, glands and viscera. Both systems differ essentially in their physiologic manifestations and pharmacologic reactions. Anatomically, the parasympathetic system (midbrain, bulbar and sacral autonomic outflow) consists of medullated nerve fibers, while the sympathetic proper is composed of nonmedullated nerves. Unstriped muscle, like striated muscle, is composed of fibrillae and sarcoplasm. The fibrillae pass from one cell to another forming a contractile network, and subserve the function of primitive movement. The sarcoplasm is concerned with primitive posture which permits the adaptation of blood vessels and hollow organs to their contents (capacity-posture).

According to my conception, both the cerebrospinal and vegetative nervous systems, as well as striped and unstriped muscle, present evidences of static and kinetic systems underlying the functions of motility.

In the phylogenesis of movement the contractile cell passes by gradual stages from the nonstriated to the striated type, heart muscle representing a transition form between the two. Posture is the dominant function of involuntary muscle which coincides with the predominance of sarcoplasm. In voluntary muscles, movement is the dominant function, and there is a corresponding differentiation of the contractile mechanism.

In both voluntary and involuntary muscles various types of muscle fiber may be recognized, representing transitions from lower to higher forms. These differences in muscle structure are in harmony with the phylogenesis of the

efferent nervous system—archeokinetic, paleokinetic and neokinetic—for the "effector" end organs which express the contractile function must also undergo changes in evolution in order to fulfil the demands of a more highly organized central nervous system.

These two systems also have important relations to symptomatology. Symptoms referable to both the kinetic and static systems may be released by the dissociations of disease. A lesion of the kinetic system causes a disorder of movement and of the static system a disorder of tonus or of posture. This principle holds for both the splanchnic and somatic systems.

The kinetic mechanisms of the somatic system may give rise to various types of convulsive manifestation, namely: kinetic types of epileptic seizures (tetanic and clonic); also chorea, athetosis, dystonia, the tremor of paralysis agitans; paramyoclonus, myokymia and fibrillary twitchings.

Related to the static mechanism are sudden postural relaxations of epilepsy, partial or general (static seizures); all forms of myotonia: cerebral, cerebellar, spinal and peripheral; also cerebellar symptoms—asynergia, dysmetria, adiadokokinesis and intention tremor.

Even in the psychic sphere evidences of a dual representation are manifest in the hyperkineses of psychic origin (convulsions, chorea, tic convulsive) and in certain postural disorders (catatonia, catalepsy). A similar interpretation may also be given to active and passive perseveration.

In the vegetative nervous system, disorders of movement are related to the kinetic system (parasympathetic). This may be expressed by hyperkinesis, for example, gastric and intestinal hypermotility.

Related to the static system or sympathetic are disorders of postural tone—conditions of atony and dilatation of the blood vessels and the hollow viscera. This conception implies, therefore, a parallelism of structure and function of the kinetic and static systems throughout the whole efferent mechanism, from its lowest to its highest levels—a duality of function which is revealed in many different fields of investigations, and which is manifested in the symptomatology of the nervous system.

Kinesthetic and Statesthetic Systems.—The sense of movement and the sense of posture are well recognized components of deep sensibility. Both of these forms of bathyesthesia are composed of sensory impressions derived from various sources, chiefly from the muscles, but also from the joints, tendons and fascia. The vestibular mechanism, as was pointed out by Sherrington, is also closely related to the proprioceptive system and plays an important rôle in the regulation of postural tone. And it is interesting to note that recent investigators (Magnus and Kleijn; Randall; Hunter) recognize the existence of a kinetic as well as of a static labyrinth in which the semicircular canals yield kinetic impulses and the otoliths static impressions. And it is not unlikely that the labyrinth has a statesthetic and kinesthetic function quite separate and distinct from one another, subserving respectively the sensory aspects of posture and of motion.

In the conception of a statesthetic and kinesthetic function I have reference more particularly to muscle sensibility (myesthesia) and its relation to the dual functions of motility. For if it be true that the efferent nervous system from its earliest development in the vegetative mechanism to its highest expression in the cerebral cortex shows evidence of a static and a kinetic mechanism, the existence of a similar division of function in the afferent sphere is a necessary corollary. For the efferent system is only one limb of the reflex arc, and where two separate physiologic systems exist subserving the function

of motility, so different in the nature of their contractile function, there must also be corresponding differences in the function and morphology of their afferent mechanisms.

One may postulate, therefore, in both skeletal and visceral muscle, the existence of special afferent systems for the transmission of sensations of movement and of posture to the central nervous mechanism. One is the kinesthetic component of muscle sensibility conveying impulses of movement (kinesthesia); the other is the statesthetic component conveying impulses underlying postural tone (statesthesia).

Anatomic Considerations.—As already mentioned, striated muscle fiber has two types of motor nerve endings, which are probably related to the kinetic and static systems of motility. One is the motor end plate which is the terminal of a medullated nerve fiber (myokinetic effector); the other is a sympathetic type of nerve ending, the terminal of a nonmedullated nerve fiber (myostatic effector). Both of these terminals are beneath the sarcolemma (hypolemmal) and therefore in direct relation with the contractile content of the muscle fiber.

In addition to these motor types of nerve endings, the investigations of Huber, Crevetin and Dogiel have shown the existence of other terminals of a sensory character in relation to the muscle fiber. These are the nerve endings of both medullated and nonmedullated nerves, and are situated outside the sarcolemma. They are found on the outer surface of the muscle fiber, the tendon and musculotendinous junction, as well as in the intermuscular connective tissue, and are evidently sensory in their function.

Dogiel, who used the methylene blue method and whose investigations were carried out on the ocular muscles (recti) of man and mammals, reached the conclusion that there are two kinds of sensory nerve endings for each muscle fiber. These are the terminals of both medullated and nonmedullated types of nerve fibers. In one form the nerve ending entwines the muscle fiber, frequently throughout its whole length. The other surrounds the end of the muscle fiber in the form of a palisade, the fiber fitting snugly into this end apparatus. Between these two typical types of sensory nerve endings of muscle fibers there are various transition forms. It is evident from these investigations that muscle fibers are well supplied with sensory nerves and nerve endings of both medullated and nonmedullated types.

It would be premature to attempt any correlation between these histologic studies and a possible kinesthetic and statesthetic function. As, however, in the effector sphere there is already considerable evidence showing that kinetic function is controlled by a medullated nerve fiber and static function by a nonmedullated nerve fiber, it is possible that a similar morphologic difference and correlation may hold for the affectors and the afferent system. For the greatest evolution and highest differentiation of motility is in the kinetic sphere, and it is therefore possible that posture function, which is automatic and secondary, is subserved in both the afferent and efferent sphere by nerve fibers of primitive nonmedullated character.

The statesthetic and kinesthetic components of muscle sensibility unite with fibers from other structures subserving the sense of movement and of posture and pass together in the spinal cord, the brain stem and the thalamocortical pathway. In conditions of disease, because of their proximity, both of these components of the proprioceptive system are usually associated with loss of the sense of movement.

Within the spinal cord, the kinesthetic and statesthetic systems pass together in the columns of Goll and Burdach to the nuclei of the posterior columns.

From these nuclei, secondary pathways pass in the corpora restiforme to the cerebellum in the interest of postural function while other fibers, both kinesthetic and statesthetic, are continued in the brain stem to their secondary terminations in the optic thalamus. The ventral and dorsal direct cerebellar tracts pass directly to the vermis cerebelli from their primary stations in the gray matter of the spinal cord.

From the optic thalamus kinesthetic and statesthetic impulses are conveyed by its commisural system to the corpus striatum for the regulation of automatic-associated movement (paleokinesis). From the optic thalamus, the kinesthetic and statesthetic systems are continued in their tertiary and final pathway to the parietal lobe of the cerebral cortex.

In addition, therefore, to these sensory structures which participate in the reflex postural and kinetic functions of the segmental nervous system, three great stations representing posture-motion groupings may be recognized; namely, in the myelencephalon (nuclei of Goll and Burdach), the diencephalon (optic thalamus) and the neoencephalon (parietal lobe).

I believe that these two sensory systems play an important rôle in the reflex production of the phenomena of reciprocal innervation, which is so striking a feature of muscular activity, namely: concomitant relaxation of the static or posture mechanism of antagonistic muscles with contraction of the agonists.

Relation of the Statesthetic and Kinesthetic Systems to Symptomatology.—A disorder of the kinesthetic system would produce a loss of the sense of movement or kinetic ataxia. A disorder of the statesthetic system would produce a loss of postural sensibility or static ataxia.

The statesthetic system is the sensory component underlying plastic tonus (statotonus), the "lengthening and shortening reactions" of muscles and other manifestations of postural tone—reflexes of posture. The kinesthetic system is the sensory component underlying the "twitch," the contractile tonus (kinetotonus) and reflexes of movement.

These two functions of the proprioceptive system are usually involved together and frequently in the same degree. In some cases, as in tabes, a more selective involvement may occur, causing kinetic ataxia, loss of tendon reflexes, with little or no loss of postural tone; on the other hand, there may be well marked hypotonia without ataxia or loss of tendon reflexes.

Involvement of the corpus restiforme by cutting off statesthetic or postural stimuli to the cerebellum will produce static ataxia. This is a pure ataxic disorder due to loss of the postural synergy of the cerebellum and is not associated with other disturbances of deep sensibility. Kinetic ataxia, on the other hand, is an incoordination of movement dependent on a loss of the afferent systems conveying kinesthetic impulses to the efferent mechanism.

ON THE OCCURRENCE OF STATIC SEIZURES IN EPILEPSY. Dr. J. Ramsay Hunt.

By "static seizures" I mean a form of epileptic seizure characterized by sudden losses of postural control. This type of epileptic manifestation I believe is related to the static system of motility, and would differentiate it from the convulsive or kinetic type of seizure which gives the characteristic imprint to the clinical picture of epilepsy. I have observed a number of patients with static seizures. The kinetic type of seizure is the common one and may result from a variety of causes, toxic, organic and emotional. The static type of seizure I have observed only in idiopathic epilepsy. It is characterized by a

sudden loss of postural control and may occur alone, as a dissociated manifestation of epilepsy. The loss of postural control is sudden and shocklike, the patient falling to the ground with abrupt violence in response to gravity. This sudden plunge or drop is characteristic and is not infrequently the cause of serious injury, especially to the face and head. It is quite different from the usual fall of the epileptic in the convulsive attacks. In one case, both patellae had been severely injured by frequent and severe drop-seizures. While the drop is sudden the postural relaxation is of short duration, the patient rising almost immediately from the ground without assistance. The fall is usually associated with transitory loss of consciousness, which may, however, be slight. In not a few instances there is scarcely any appreciable obstruction of consciousness. The fall is usually forward and is associated with sudden relaxation or "giving way" of the lower extremities. As a rule there are no convulsive manifestations, although the two varieties of attack may be combined. The postural relaxation is more or less general, the patient falling in a heap from complete loss of postural control.

Contrasted with these more or less general types of postural relaxation, these patients often show a more limited or local form of postural relaxation, which may be quite circumscribed in character and distribution, and associated with myoclonic jerks or starts. Such myoclonic manifestations are not uncommon in the early morning in cases of epilepsy, particularly on arising, and are often relieved or lessened by the recumbent posture. They are characterized by sudden muscular jerks or starts, often bilateral, which may affect the arms, trunk, head or legs. Usually there is only a single muscular contraction, although these may follow one another in rapid succession. Patients during the period of myoclonia are liable to drop objects held in the hand and typical general static seizures in this group of cases, from a more general postural relaxation, are not uncommon.

While in the present state of our knowledge one cannot assert positively that these myoclonic manifestations are exclusively related to the static system, it is my belief that a close relationship exists, and that the myoclonic jerk or start is often only a secondary or compensatory kinetic manifestation in response to sudden localized relaxation in the posture sphere. From experimental evidence, some posture relaxation precedes or accompanies nearly every form of cortical movement, so it is possible that both of these elements may play a rôle in these minor motor manifestations of epilepsy. The typical drop-seizures are by no means common, and up to the present time my experience is limited to ten cases. When these attacks are present they tend to recur with a certain degree of regularity and persistence.

A few months ago, under the title "Dyssynergia cerebellaris myoclonica," I reported a group of cases in which the patients had symptoms of cerebellar disease associated with myoclonus epilepsy. In several of these patients typical static or drop-seizures were observed. In one case examination of the central nervous system revealed a primary atrophy of the cells of the dentate nucleus and its efferent system in the superior cerebellar peduncles. This lesion causes a break in the static or posture system and may have some relation to the myoclonia.

The neostatic system, I believe, arises in close relation to the neokinetic area of the rolandic region. It then descends in the anterior limb of the internal capsule, the mesial portion of the crus cerebri to the pons, where it terminates in relation to the ventral nuclei of the pons varolii. This corresponds to the frontopontile tract of neuro-anatomy. From the pons the fibers cross to the

opposite cerebellar hemisphere and then descend by way of the dentate rubrospinal and other systems to the sarcoplasm of muscle, the function of which is fixation of the muscle fiber in terms of posture. The static seizure, I believe, is related to a disorder of this mechanism.

Hering and Sherrington showed clearly that postural inhibition and muscular contraction could be elicited by electrical excitation of the cerebral cortex and that there exists a form of coordinate innervation in which the relaxation of one group of muscles occurs as an accompaniment of the active contraction of another set.

The experiments of Sherrington were carried out on cats and monkeys. He was able to show that stimulation of the appropriate center, e. g., that presiding over extension of the elbow, produced an immediate relaxation of the biceps, together with active contraction of the triceps. As soon as the stimulation is discontinued the arm returns to its previous posture of flexion. By weakening the faradic current, relaxation can in many instances be induced without any obvious contraction of the opposed muscles. The relaxation seems to occur quite synchronously with or sometimes a little prior to the contraction of the opposite group. The points of cerebral cortex from which relaxation and contraction of a particular muscle, e. g., the biceps brachii, can be evoked respectively are distinct from one another, and often even in a small monkey lie more than a centimeter apart. Besides, therefore, a localization for muscles according to their contraction, there is also a cortical localization different in scheme and capable of demarcation by observations with relaxations as index. It is interesting to note that Sherrington also obtained relaxation of certain muscles by stimulation of various points on the cross section of the internal capsule.

Sherrington also confirmed these results by cortical stimulation in decerebrate rigidity. After ablation of one cerebral hemisphere a homonymous extensor rigidity develops which presents an opportunity for examination of the sphere of excitation of the cortex on the extensor muscles of the crossed elbow and knee. He found in the rolandic region of the monkeys a cortical area which gives markedly and forthwith inhibition of the contraction of the extensors of the elbow, and another cortical area which similarly when excited inhibits the contraction of the extensor of the knee.

Weed, in an elaborate study of decerebrate rigidity, has still further clarified this subject. Weed found that inhibition of the extensor spasm of decerebrate rigidity could be obtained from the motor cortex of the rolandic area, from the mesial anterior portion of the internal capsule, from the mesial one-sixth of the crus cerebri, from the pons and from the anterior portion of the superior vermis.

In a later study Cobb, Bailey and Holtz also investigated the genesis and inhibition of extensor rigidity in cats. They found that electrical stimulation of the cortex of the anterior lobe of the cerebellum produced an inhibition of the rigidity in the ipsolateral muscles. Finally the anterior lobe was removed exposing the dentate rubral tracts, stimulation of which produced an even more marked inhibition of the extensor rigidity.

Therefore, on the basis of clinical observation and experimental data, I would postulate the existence of a type of epileptic manifestation characterized by sudden losses of postural control. I believe that these are referable to a loss of static control and may be regarded as static seizure, in contrast to the convulsive manifestations which are essentially kinetic in origin.

DISCUSSION

DR. L. PIERCE CLARK: Since knowing Dr. Hunt's view relative to static seizures in epilepsy, I have been keen to find them, but I have not seen such attacks. The report of a pure static seizure without slight preceding tonic spasm I believe is due to faulty observation. We all know, however, that the tonic, clonic and simple paralytic phases of an epileptic seizure are subject to wide fluctuations in their proportional relations in a given seizure and in different persons. While any particular seizure may be preponderantly static in type, I believe in the dictum of Hughlings Jackson that some degree of muscular spasm invariably occurs in every epileptic attack and that if studied closely the tonic element will be disclosed at the inception of supposedly pure static seizures.

DR. SMITH ELY JELLIFFE: I think Dr. Hunt is right in the possibility of the static type of loss of function. He can call it epilepsy if he cares to do so. There is a sudden failure of the geotropic mechanism. Whether his analysis of the pathways is valid or not, I cannot say. Dr. Hunt speaks of the function of the otoliths. I think that the inertia stimulus is an important factor. We must have some function of inertia. The planet we are on is being whirled in space at an enormous speed and we and our whole system are carried at a tremendous rate, so that inertia becomes an important force. The recent work of Magnus, Winkler, Kleijn and the whole Dutch school has emphasized the function of vestibular mechanism as handling inertia and geotropic stimuli.

DR. C. L. DANA: I have somehow a feeling that the phenomena of mental and neural function cannot be altogether explained by a system of more and more complicated and integrated mechanisms. There must be an explanation of what balances the integrations and how the machines still work when most of the machine is gone, as in people without any labyrinth and little cerebellum and no cervical sympathetic system to speak of.

In regard to the static epileptic seizures I think Dr. Hunt is right. Some years ago I reported such a case which seemed to correspond with the old term of "falling sickness." My patient, a boy, would "crumple up" in the street or at home with instantaneous relaxation of all his muscles. There might have been an unobservable period of muscular tonus first, but I could not see it. The boy simply dropped and arose immediately. The case was reported as true "falling sickness." I have seen several others since, and Dr. Hunt has described the characteristic phenomena correctly.

DR. I. ABRAHAMSON: We must be careful in assuming that agonist and antagonist always act in the manner described. In encephalitis I have seen several cases in which the biceps and triceps contracted at the same instant. We should first show static seizures in jacksonian epilepsy. The crumpling up may be an expression of more or less sudden and complete loss of consciousness. Gyratory, propulsive or retropulsive seizures, falling to either side or forward and backward like a falling tree, would be more suggestive of static attacks than a mere crumpling up.

DR. RAMSAY HUNT (closing): I agree with Dr. Clark that in some cases of sudden postural relaxation (static seizures) it is difficult to say positively that there is not some perturbation of function in the kinetic sphere. That, however, does not invalidate the essential features of my hypothesis. It is a refinement of diagnosis and has more of a physiologic than practical interest. It reopens the question of postural relaxation in reciprocal innervation, a question which Sherrington initiated several years ago.

THE NATURE OF ESSENTIAL EPILEPSY. Dr. L. Pierce Clark.

The epilepsies, when stripped of all their organic and symptomatic types, leave a large group of epileptic patients who, apparently, in spite of any obvious cause, still have periodic convulsions, and to this group is applied the term essential epilepsy. It has been found that every such person possesses a primary defect in the instinctive life, called the epileptic constitution. The glaring clinical manifestation of such a personality is its crude form of egotism, possessing a correlate of extreme supersensitiveness and an emotional poverty as a part of the defective developing character. We then postulate that social and life adaptations in such persons cannot be met without enormous stress, and the varied life issues entailing the latter precipitate epileptic reactions. We therefore look on the convulsion as a break in the life demand for adaptation, and the nature of the convulsion as a protective and regressive phenomenon. The more severe and frequent it is, the deeper the regression. We have thoroughly detailed clinical material to substantiate this thesis, namely, that essential epilepsy is really based on the defective primary endowment, the epileptic constitution.

DISCUSSION

Dr. Smith Ely Jelliffe: I see no contradiction between the terms functional and organic. Every function has to work through an organ, so that to separate functional and organic in the deep philosophic sense is impossible. Where does Dr. Clark put the separation of functional types from organic types? He says he has pushed out every case that can be explained, such as those caused by bony spiculae, inflammation, tumor, etc., and that leaves a residue of cases that he calls essential epilepsy. In these cases he finds a certain type of constitution which, he says, is based on defective development. What does he mean by defective development? If he takes up psychogenic epilepsy, does he mean that the development is mental? Are "psychogenic" and "mental" synonymous terms? He has not made clear what he means by defective development. If he means by "psychogenic" the function of handling symbols, I can understand what he means by an essential epilepsy. The symbol functions at a high energy potential. When captured by the body it must be transformed and the energy redelivered. Symbolic delivery is the most essential and dynamic in the behavior mechanism. A psychogenic epilepsy, then, is one in which the symbolic functioning is defective.

Dr. Bernard Sachs: Dr. Clark has used a number of relatively new terms and expressions, but I should like to inquire in an entirely friendly way whether in his own mind he has gone beyond the old postulate that the epileptic patient has a predisposition to this special form of disease; or that his brain is subject to these peculiar discharges and anything going wrong in his body will aggravate them.

Dr. M. Osnato: I believe that Dr. Clark stated that the delirium in petit mal was of the functional type because its content dealt with the everyday life experience of the individual. I do not think that is any criterion. All delirial productions deal with ordinary life experiences, with what is in the consciousness. Therefore this does not prove that the delirium in petit mal is functional and not organic. I think there is danger in laying too much stress on the makeup and personality as determining the mental disease from which the patient suffers. The three points which Dr. Clark emphasizes—egocentricity, emotional poverty and hypersensitiveness—are not different from those found in the personality of patients with dementia praecox. The type of reaction is

influenced by the makeup but not by the disease itself. Dr. Clark said that a tyro can pick out the epilepsy makeup but that the epileptic patient may have minor degrees of the same attributes and that only the carefully trained physician can bring these out. The same qualities that are supposed to belong to the epileptic makeup are found in the characters of many people. Why should the mechanism be different in epilepsy if it is of psychogenic origin? Why should the psychologic mechanism work differently in epilepsy and express itself in convulsive manifestations which are objectively and by various chemical and other means so readily differentiated from the convulsive manifestations of hysteria?

Dr. I. J. Sands: Many points mentioned by Dr. Clark do not agree with my experience. In the first place, I take issue with him when he says that there is a primary defect in the instinctive life of the epileptic patient. When one thinks of the instinctive life of a person as ordinarily described by such men as Woodworth, McDougall, or Watson, representatives of the different recognized schools of psychology, it is difficult to find any real defect in the instinctive reaction of the epileptic. In fact, the instincts are well developed in him. In the elaboration of this theoretical defect in the instinctive life of the epileptic patient, Dr. Clark mentions the egotism, supersensitiveness and emotional poverty of the epileptic. It is true that one encounters these characteristics in epileptic patients, but their genesis is not to be found in instinctive defects. They are to be best explained as resulting from defensive mechanisms induced by an inferiority complex. The epileptic patient is fully aware of his handicap in life. This induces in him a feeling of inferiority. He is quick to sense any danger. He is very sensitive, or supersensitive, in order to detect any danger in the environment that may mean destruction to him. He is quick to detect any menacing situation that may come up in his sphere of activity. Such a personality is very unpleasant and is not conducive toward a frank and open relationship with one's neighbors. The epileptic patient becomes more or less of a social outcast, and for this state of affairs he does not seek an explanation in his own personality but in those of the people with whom he comes in contact. This in turn tends to make him egotistical as he is constantly finding imaginary faults in those with whom he comes in contact in his effort at rationalizing his exclusion from social organizations. The emotional poverty is partly explained as a result of the traumas caused by the convulsions, and partly as a sequence to the supersensitiveness and egotism of the person. Furthermore, we must not lose sight of the fact that there are quite a few epileptic patients who are very meek, altruistic and tolerant, and this too as a result of their inferiority complex, hoping to counteract their handicap by assuming such attitudes in their dealings with their fellowmen.

In the second place, I never could fully understand Dr. Clark's contention that the seizure is a mode of escape from an unpleasant situation. There is nothing more unpleasant and more serious that occurs in the life cycle of the epileptic patient than the convulsion. Each seizure leaves an indellible mark on the personality.

In the third place, I beg to take issue with Dr. Clark on his characterization of the epileptic delirium as a psychogenic delirium. At Bellevue, we are in a most favorable position to study the various forms of delirium. There is no doubt in my mind that the delirium associated with epilepsy is of organic nature. It resembles most closely the delirium resulting from cerebral

trauma; in fact, I believe that they are identical. I can never differentiate the two except from the history of the case. I believe that epilepsy is the resultant of some cerebral insult, gross or molecular structural alteration in the brains of the person, which is escaping detection through the present methods of investigations. The convulsions are responses to stimuli, endogenous, exogenous or psychogenic, sent to the highly sensitive brain of the patient. If the assumption that there are either gross or molecular changes in the brains of the epileptic patient be true, one might expect to find the type of epileptic patient described by Dr. Hunt. Clinically I have seen such a type coming into the psychopathic wards of Bellevue. It is difficult to prophesy where the locus for the ultimate solution of the genesis of epilepsy lies. More facts are needed to evaluate the effect of disease in the mother on the brain of the developing fetus. Furthermore, we need more data in regard to the influence of mild infections on the developing and growing brain during infancy.

Lastly, I cannot understand why Dr. Clark persistently asserts that phenobarbital has a detrimental effect on the patient, asserting that it causes deterioration. This is entirely different from the statements made in reports that are now swelling the literature. I have used phenobarbital extensively, not only in the sane epileptic patient but also in the psychotic one. I have never had any untoward results. Not only has it failed to cause deterioration, using the word in its restricted psychiatric sense, but it has helped to clear the psychotic episode and has prevented deterioration. In only one case have I seen bad results from phenobarbital, and that was in a young epileptic patient who had been receiving 5 grains (0.32 gm.) of phenobarbital three times a day over a period of ten weeks. That patient finally presented the typical signs of so-called "barbital poisoning." This was not the fault of the drug but of the physician who had used it improperly.

Dr. J. Ramsay Hunt: I should like to ask Dr. Clark his reasons for assuming that all cases of idiopathic epilepsy are psychogenetic. Epilepsy is a symptomatic manifestation which has definite associations with organic disease and intoxication of various kinds.

It is only in recent years that the pathology of paralysis agitans has been placed on a fairly firm basis. Before this many theories were advanced which are now no longer seriously considered. It seems that we are still in this period with regard to epilepsy. Let us hope that before many years have elapsed the epilepsies will also be placed on a more secure pathologic foundation, and then many of the theories which now engage investigators will have only an historical interest.

Dr. Israel Strauss: Let us suppose a boy 5 years of age has a history of convulsive seizures. We examine him and find nothing wrong organically. The metabolic, endocrinological and focal infection studies reveal nothing unusual. Such a case, according to Dr. Clark, is idiopathic. We must then seek for the attributes of the epileptic constitution which, as Dr. Osnato has pointed out, are present in a good many of us. This makeup has led to a conflict in adaptation and as a result of it the patient has developed epileptic convulsions. This would not appear to be a satisfactory and pleasurable solution for most persons. If now we find no organic cause for the epilepsy in this boy and no evidence of the epileptic constitution, what form of epilepsy has he? Some years ago Dr. Clark would have found the explanation in the attempt of the child to go back to the uterus of the mother, this leading it to assume the fetal position in the tonic state of the convulsion. I cannot see

the rationale of this. In seeking the solution of the problem of epilepsy we shall have to deal with fundamental processes in the life of the organism. We cannot solve it by studying the human being. I believe we must study lower forms of life from the point of cell metabolism and activity, for there lies the secret of what produces this tremendous change in the human brain.

Dr. C. P. Oberndorff: I have seen one interesting case of the "crumpling up" type of epilepsy mentioned by Dr. Hunt, in a young man of 26, 6 feet 2 inches (187.96 cm.) tall, with a prominent chin but no other signs of acromegaly. He was playing golf when he suddenly dropped. Since then he has had three or four attacks; in one he was cut severely by striking his face. In addition, he has shown a tendency to somnolence. He falls asleep in the train or even while talking or dancing. He is at present confronted with an emotional conflict in that he is Protestant and the girl he wants to marry is Catholic. On leaving the girl's house he has an attack of epilepsy or somnolence, so that he does not know what he is doing. While asleep he walked through a glass door and was badly cut. I did not wish to use psychanalysis in this case but hypnotized him with great facility. He tells me the mental state in hypnosis is different from that in the epileptic form attacks. He has a normal sella turcica, no increased sugar tolerance, or acidosity; but I think he has pituitary disturbance. Two views have been taken of his disorder: it is glandular or psychogenic. I thought it pituitary.

Dr. P. R. Lehrman: The difference in the views expressed impresses me as due to the difference in the mode of approach to this problem. The organic approach has so far yielded little. I have closely followed Dr. Clark's studies and in several instances have been convinced of the truth of his observations. I know of one man, aged 35, a Seventh Day Adventist minister, who has petit mal attacks, whose case was diagnosed as essential epilepsy after careful study at the Vanderbilt Clinic. Phenobarbital did not help him. I became interested in his utterances while he had an attack. He would then invariably repeat a Catholic prayer. This was a reversion to early training. The patient became a convert at the age of 17 out of protest at his tyrannical father. The relationship of such a personality reaction to religious conversion is significant. I believe that the more we study the problem of epilepsy from the point of view of personality and are willing to hear what the patient says, voluntarily and while associating, the quicker we shall be able to evaluate properly the method that Dr. Clark is using in his studies.

Dr. L. Pierce Clark (closing): I believe that essential epilepsy is at the bottom organic, or better, constitutional. The defect at inception of the disorder is not that form of brain lesion which should properly be classed as the symptomatic pathology. This primary defect is now and perhaps may always remain nondemonstrable by our present methods of study. The defect is shown in the imperfect development of the instincts of the epileptic person as a whole and not in any one of his special functions of brain structure. My method of approach may seemingly be a regressive one and not in accord with what we may term the mechanistic studies of the immediate past. It, however, really rests on the method perhaps first inaugurated by Hippocratic studies in epilepsy. It is essentially the psychobiologic one, which includes the inheritance, the present makeup, and the environmental factor. It may seemingly neglect the meticulous exactness of individual study of special parts of the brain because it maintains that we may not wrench such a function from its cooperative functioning with all other parts of the brain and the whole body as well.

My fundamental postulate is that epilepsy in its entirety is a life reaction disorder and must be studied on this basis. It is basically a dynamic approach to the evaluation of the essential defects of the whole organism. It has its specific mechanistic defects in brain structure cells, tracts and neural envelops; it has metabolic and katabolic disorders in the bodily tissues. These are all but correlates of the fundamental defects of the whole epileptic person. One may say, if we take a summation of all the isolated disordered functions of the whole brain, may we not build up a comprehensive picture of epilepsy? No. The dilemma then may be illustrated by MacCurdy's example in chemistry: Sodium and chlorin when united produce common table salt whose qualities can in no wise be postulated from a consideration of the single elements before their chemical union. So there comes into being a something unpredictable from the summation of the several organs and functions of the body. The biologic study of different disease processes, such as epilepsy, is absolutely essential to get at the broad fact of the dynamic etiology of the disorder. This manner of approach by modern scientific principles is relatively new and has far to go before neuropsychiatric problems will yield final and satisfactory results. What I have so far brought forward in my studies is really not radically new in any one of its tenets. It is the grouping and the more exact study of the formerly loose designation of predisposition that brings a seeming novelty to the formulation, and the great importance I place on the dynamics of the makeup in determining the disease as such. In regard to the study of epileptic delirium, one perhaps may not designate whether it is really psychogenic in character, from the content alone, but if one is able to show by after-analysis that the conflict revealed in delirium is removable by psychologic efforts it shows for practical purposes that the factors at work are probably psychogenic. If phenobarbital really did anything more than repress the fit and cause the patient to live at a lower level of life adaptation, Dr. Strauss' remarks on this part of our subject would be more pertinent. Sedatives alone can only work harm in the disease process as a whole in the long run. Even studies on the epileptic personality and makeup are not final, exclusive and inclusive; but we are on the right road and such studies properly correlated, with coincident changes in all bodily tissues, will give us new understanding of the disease process of epilepsy. The line of study is not dissimilar to many others in the more advanced study of the psychoses. The need of united mechanistic and psychobiologic study of our problem is obvious and each will make its value felt in the final solution.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY*

Regular Monthly Meeting, April 20, 1922

DR. F. H. PACKARD, *President, in the Chair*

SOME OBSERVATIONS ON THE CHEMISTRY OF EPINEPHRIN. DR. J. C. WHITEHORN (by invitation).

1. Epinephrin in strong concentrations gives a violaceous red color with the reagents customarily employed in testing for tryptophan or its derivatives; that is, concentrated sulphuric acid and glyoxylic acid, vanillin or formaldehyd.

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* Communications from members of the staff of McLean Hospital, Waverley, Mass.

This suggests a possible metabolic relation between epinephrin and tryptophan, and therefore makes desirable epinephrin studies in such conditions as pellagra.

2. Neutral formaldehyd, which inactivates epinephrin physiologically, has the chemical effect of almost, if not quite, destroying the basic character of epinephrin.

3. A new oxidative color reaction by means of silver peroxid has been found to be several times as delicate as the Ewins reaction. The silver peroxid is prepared by mixing equal volumes of one hundredth normal silver nitrate and 2 per cent. potassium persulphate. When 0.25 c.c. of this freshly prepared suspension and about 3 c.c. of dilute epinephrin solution are mixed, the addition of from three to five drops of ammonia water gives a pink color, which increases in intensity for four or five minutes and then begins to fade.

A PSYCHIATRIC INDEX FOR FACILITATING THE STUDY OF PREVIOUS RECORDS. Dr. S. M. Bunker (by invitation).

The object of the psychiatric index is to promote a larger use of the psychiatric hospital records. The index essentially consists of about one hundred psychiatric symptoms selected as chiefly characteristic of the more common psychoses recognized today. Graph paper gives an opportunity to check in chronologic sequence the presence or absence of these symptoms in the development of any particular case. Case record systems have been developed at the expense of considerable labor and money in general hospitals and case records in general hospitals are used much more frequently than in psychiatric hospitals. The symptom index has been found useful at the Danvers and Rhode Island State Hospitals. The present index has been devised to serve as a key to records at the McLean Hospital. It shows the trend of the psychoses and stimulates interest in the chronic case. It is made elastic by providing for inclusion of symptoms peculiar to any particular case, thus aiding in the analysis of the individual case. Card index files are made from the symptom index which is filed with the case record. The consistent use of the symptom index would present several worth while possibilities: 1. It offers the basis for a dictionary of psychiatry. 2. It provides a quick and scientific method of presenting cases of like symptomatology at staff conferences. 3. It accepts Kraepelinian terms at their face value, thereby laying a fair basis for criticism of the whole Kraepelinian school. 4. It encourages students of psychiatry and psychology at our universities to consult original records rather than to depend on textbook theories. It is not a statistical study, but is designed to unlock the accumulated records of the past and make them accessible to the student of today.

DISCUSSION

Dr. H. I. Gosline: I have been working on this subject since 1914 and am familiar with the index used at Danvers State Hospital. When I was at Danvers the first record was made by the assistant superintendent before the patient was brought to the clinic. At the Rhode Island State Hospital at Howard it is made while the patient is in the clinic. I consider the Danvers method preferable because there is more time to do it.

The clinic director should have charge of the records. In some hospitals the same person is clinic director as well as pathologist, but this is unsatisfactory unless he has numerous assistants. Among these assistants should be a statistician, for a hospital of 1,400 patients.

Either we do not know enough about psychology or we psychiatrists have not used what is known by the psychologists. In making the new classification at Howard we have tried to arrange the psychopathologic symptoms as to whether the primary mode of appearance is in the perceptional or ideational sphere, the sphere of inner states or the sphere of activities. We have even tried to decide whether the symptom is in the perception of concrete objects, in the perception of time or space or of measuring; whether the ideas are defective; and whether the defect is of memory ideas, of imaginative ideas or of general ideas. Another advance that might be made is to separate the physical, mental and social aspect in recording histories. We are trying to find out more about the mental and social characteristics of the forbears as well as of the patient so that when the summaries are made out there may be included the mental history with the mental examination and the physical history with the physical examination. We may have a summary of the social condition but not a social examination. Perhaps in the future there may be some way of making a social examination. In approaching the social side I have found McDougall's "Social Psychology" most serviceable. Münsterberg's arrangement in his "Psychology, General and Applied," has made it possible to group the mental symptoms according to psychologic categories.

Lastly, I would criticize the grouping together of hallucinations and illusions. They are different and probably fundamentally so.

REPORT OF A CASE OF ADDISON'S DISEASE WITH PSYCHOSIS. K. J. TILLOTSON (by invitation).

A single woman, 50 years of age, a housekeeper, developed Addison's disease beginning in September, 1920. The case was typical. One year following the onset of this disease a depression set in which had considerable involutional coloring. This progressed to an acute hallucinatory episode which was followed by months of a confused, inaccessible period ending with sudden and practically complete disappearance of all psychotic symptoms, but with slight associated physical improvement. The case is reported because the literature contains no description of the association of Addison's disease with psychosis.

REACTION TIME AS AN INDICATOR OF EMOTIONAL DISTURBANCES IN MANIC-DEPRESSIVE INSANITY. DR. HELGE LUNDHOLM.

Simple reaction times to sound stimuli were taken for twelve patients suffering from manic-depressive insanity, the readings being made twice a week for periods of four to seven months. Ten reaction times were taken every day of experimentation, and averages and standard deviations calculated. Clinical observations of the patient were carefully recorded independent of the laboratory study. The experiments show that an increase of the averages and standard deviations of a single day and also an increase of the averages and standard deviations for a period occurred as soon as a patient went into manic excitement. In four out of six instances the changes in the reaction times appeared before any noticeable change in the conduct on the ward, and consequently, the onset of the excitement could be predicted by the laboratory records. In a few cases of hebephrenic dementia praecox the characteristic finding was an absence of congruity between the clinical and the laboratory records. The latter indicated disturbance of one or the other kind, while the conduct on the ward was reported to be fairly normal. It is suggestive that

such a discongruity was found only in the introverted praecox personality type. The depressed patients were found to give a typical performance, the main characteristic of which was a considerable steadiness. In certain instances, however, there was an increase of averages and standard deviations, and this was always due to the fact that one or sometimes two reaction times of ten were much longer than the rest. These patients consequently gave either a high and narrow frequency curve approaching the normal, or gave the majority of the readings such a high curve while one or two times were at considerable distance from the rest on the abscissa. In opposition to this the frequency curve of a manic was always broad and low. Readings from patients in agitated states always gave frequency curves of the manic types, independent of the mood.

FURTHER OBSERVATIONS ON A RATING SCHEME FOR CONDUCT. DR. JAMES S. PLANT.

One year ago this scheme was reported before the Society in embryonic form. With a year's experience at this hospital and certain briefer trials at other hospitals certain questions arising at the beginning can at this time be answered. Pearson's coefficients were calculated for all of the categories. Certain difficulties are still existent which we hope to overcome. The standard deviation of the mental categories is of great interest and probably some importance. I believe the scheme has great practical value.

Regular Meeting, May 18, 1922

F. H. PACKARD, M.D., *President, in the Chair*

THE RELATION OF FEEBLEMINDEDNESS TO A CRIMINAL CAREER. A. W. STEARNS.

Four men were standing on a street corner, and at the suggestion of one of them they stole an automobile and started driving about. They were stopped by a policeman, and one of the men shot him. They were all convicted of assault with intent to kill and given from seven to nine years in the state prison.

The first man, Y., aged 20, had remained in school until he was 12, reaching the second grade. He had more or less trouble of a minor nature with the police and the truant officer. He was accused of burglary and sent to the Lyman School. A short time after leaving there, he was arrested four times for burglary, larceny, disorderly conduct and attempted larceny, was sent back to the Lyman School, ran away after nine months, and was committed to the school for the feebleminded at Waverly, where his mental age was said to be 8 years, and he was called definitely feebleminded. In 1918, he was again at large. He was arrested for burglary and served eight months at the Concord Reformatory. Shortly after leaving there, he became a member of this group. While at Concord, he became acquainted with D. and M., two members of the party. The fourth member he had known as a boy, and they had been associates for a number of years.

The second member, C., aged 19, reached the seventh grade in school, and had worked for two years as clerk in a store. In 1914, he was arrested for disturbing the peace; in 1915, for larceny, and in 1917 for larceny, when he was sent to Shirley for a short while. Later, he was arrested and sent back for eleven months. In 1919, he was arrested for stealing an automobile on two occasions, and was sent for a year to Concord Reformatory. At Concord, his mental age was found to be 9.4 years and he was classified as not feebleminded. While at Concord, he became acquainted with D. and Y. When at Shirley he became a friend of M.

M. went a year and a half to high school. He did well and was not troublesome until he was 16. At that time, he was arrested for stealing an automobile, and was sentenced, but the sentence was suspended. In 1918, he was again arrested for stealing an automobile, and was sentenced to Shirley, but the case was appealed. In 1919, he was arrested on three counts for stealing automobiles, and again the case was filed. He was again arrested on the same charge and was sent to Shirley, where he became acquainted with C. In 1919, he went to Concord Reformatory, where he spent nearly nine months. His intellectual level was 16, his intelligence quotient 1. He was classified as a responsible offender.

D. went two years to high school and did well, but had had trouble previously. In 1912 he was arrested for larceny, in 1915 for assault and battery, in 1916 for breaking and entering, in 1918 for drunkenness and in 1918 for larceny. He was sent to Shirley, where he spent ten weeks and became acquainted with C. In 1919, he was again arrested for stealing goods and was sent to Massachusetts Reformatory, where he became acquainted with M. and Y.

Of these four persons, one was definitely feebleminded, and one on the ragged edge and according to the scale feebleminded, but Dr. Fernald's opinion was that he was a borderline case. Two were not feebleminded but according to the tests a trifle superior. The two superior ones had good homes. Y had a broken home and he had grown up on the street. C. had an unsatisfactory home and a drinking father.

When studies were made of prison populations a few years ago and a considerable percentage were found to be feebleminded, we all felt that a great advance had been made in the study of criminology; but I find myself less and less satisfied with feeblemindedness as a sole cause of criminality. These four persons had almost identical careers. Is it fair to say that because one is feebleminded his career is explained, or should we search farther? This particular gang had been organized on the basis of common experience. Making a combination of hedonism and the gregarious instinct and judging individuals by elements of conduct, on the basis of a very strong tendency to conform to the customs, habits and conduct of associates, is there not a stronger argument in favor of factors other than the feeblemindedness determining the criminality in these cases? A short while ago, it was found that of a certain prison population 28 per cent. were left handed, and that fact could be used as an argument that left handedness was a cause of a criminal career. The group in question is a typical one. It is common to have from three to half a dozen young men arrested for the same offense, showing all degrees of intellectual attainment. In the state prison, about 20 per cent. are men born in Italy who are convicted of second degree murder. One case in particular is that of a man who was sitting about a card table with other men. They had had a drink or two, a quarrel began and he was expected to fight. He pleaded with

his associates that he had a family and he did not want to fight, but by public opinion he was forced to; he killed his adversary and was given a life sentence. He had conformed to the standards of his social station. Is it not fair to assume that the imbecile's career is at least as much dependent on his associates and on his experience as on his intellectual inferiority? The pressure to a criminal career is felt out of proportion by the feebleminded, yet feeblemindedness in itself is not an adequate and complete explanation of a criminal career. The elements in a criminal career must be looked for quite beyond feeblemindedness, and they may be found in sociologic study oftentimes as much as in psychiatric study.

DISCUSSION

DR. F. L. WELLS: The finding in delinquent cases is not necessarily one of defective intelligence. As in the psychotic cases, we may find a lowering of the intelligence level, but at the same time many cases are at and above the normal average. Some interesting studies have been made by Murchison of the college graduate as an inmate of penitentiaries. His work was done in Ohio, and he found that the proportion of college graduates in prison was somewhat larger than college graduates in the general population of the state.

DR. EDWARD B. LANE: One difficulty in discussing this subject is that feeblemindedness is often described as an entity. It is not an entity. It is recognized in certain persons as a defect, and these defects are various. The majority of feebleminded persons are not criminal in their tendencies. A few years ago, the social workers ran off at a tangent and sought to prove that every immoral woman was feebleminded. A certain school teacher who had chosen to lead an easy life was kept under watch for a year as a feebleminded person, but after careful examination I could find no reason for considering her such. There is no reason to presume that because a person is feebleminded she is immoral or vice versa. But the practical point is that society has to be protected. This is the business of courts and of physicians. We used to hear of moral imbecility. The term is not used often now, but it describes the condition of people who all their lives, until 60 years of age or more, can never control their selfish desires and will never allow consideration of the rights of others to restrain them. A speaker has mentioned the knowledge of right and wrong test; these defectives are very keen to detect any attack on their own rights and make loud protests of the wrong done them.

DR. J. A. HOUSTON: The majority of repeated offenders that we are examining now in the courts, although perhaps not intellectually feebleminded, do show a marked defect. They can be picked out as being a little peculiar and different. They are defective in many ways; deficient in judgment, defective in their sociologic and moral sense. They have no regard for the rights of others; they are selfish and not amenable to fear of punishment nor susceptible to the rewards of well-doing. There is a defect, though it may not be a defect of intellect.

The state should take control of the care of its defectives. Care of the criminal class should be taken out of county control. Dr. Stearns has spoken of the four men becoming acquainted in three different institutions. In the county jails, there is too much opportunity for criminals to associate with each other. The last group which I examined had become acquainted at the state prison. There they had met a third person who was there for breaking and entering, who told them the circumstances of his burglary. Prior to their

release, they had formed a plan, and when free they carried it out, breaking into two stores. They were arrested and while awaiting trial they were associated with eight or ten others in jail, where they had nothing to do to occupy their time except to read, play games, tell stories and recount their experiences. These men had arranged their stories to be told at their coming trial so that they would all agree. It seems that the state might handle such cases more rationally than is being done by the counties.

DR. KARL BOWMAN: Our experience at the Boston Psychopathic Hospital shows definitely that a large percentage of criminals are not feebleminded. Feeblemindedness is not the real basis of criminality. Our examination has not helped us to understand why these individuals are criminal. We can simply say that they have taken up a certain way of behaving. Dr. Tredgold, in the last number of the *Journal of Neurology and Psychopathology*, endeavors to explain this condition as an arrest of development. He has some very helpful ideas. One reason why the feebleminded person tends to become criminal is emphasized by Dr. Goddard in his latest book. An individual of less than 12 years is unable to grasp abstract ideas, and therefore, the inculcating of abstract ideas of justice, honesty, etc., cannot be successfully accomplished by ordinary methods.

DR. A. W. STEARNS, closing: Judging from these four cases, it seems highly improper to use a criminal career as an important factor in differential diagnosis, as is so frequently done. The social conduct is given too important a place in the diagnosis of feeblemindedness.

THE CEREBROSPINAL FLUID IN JAUNDICE. HUGO MELLA.

What happens to the cerebrospinal fluid in those patients who have been treated with arsphenamin who develop jaundice? In 1912, Mestrazat studied the spinal fluid in four cases of icterus and only one patient gave a response to the bile test, and that was questionable. The other patients all gave negative tests, but still the fluid was yellow.

Of five patients whom we have had at the Long Island Hospital, Boston, one had been treated with arsphenamin for seven weeks. He had a negative blood reaction and a negative spinal fluid. After his last treatment, he developed marked jaundice. On testing his spinal fluid, we found the Wassermann reaction negative, the precipitation about normal, the alcohol test positive, ammonium sulphate test negative, colloidal gold not affected and the usual tests for bile on the urine positive. The spinal fluid was canary yellow. Tests applied for bile pigment and bile salts were negative. The second patient had a positive blood reaction. One week after his last treatment with arsphenamin, he developed a severe jaundice. In addition, there was a question of cord compression as he had a fracture of the first and second cervical vertebrae. There was evidence of mechanical block, but on lumbar puncture there was no clinical evidence of cord compression or subarachnoid block. If there is cord compression, there should, theoretically, be massive coagulation, and he had no massive coagulation. The spinal fluid Wassermann reaction was negative, but the gold test ran 3333211. Whether this curve was due to an old infection or whether the foreign body in the fluid from the jaundice produced this result, I cannot say. His bile tests, however, were negative. Of five other patients, practically the same facts held true. The fifth patient had received no treatment. This patient had a carcinoma of the pancreas and developed

marked jaundice. The spinal fluid was greenish yellow, but not the typical canary yellow that is seen in jaundice following arsphenamin therapy. That fluid also failed to respond to any of the bile tests. The question is, why are these fluids colored? Apparently, the color is not due to bile pigments, bile acids or bile salts. It might be possible that the meninges are colored and that the fluid took its color from them. Xanthochromia will certainly still bear investigation.

CEREBROSPINAL FLUID IN JAUNDICE*

Case	Blood Wassermann Reaction	Weeks After Arsphenamin	Fluid Pressure	Color	Cells	Alcohol	Ammonium Sulphate	Colloidal Gold	Diagnosis	Spinal Fluid Wassermann Reaction	Degree of Jaundice
1	—	7	120	Canary yellow	0	+	0	— — ± 1 ± — — — — —	Arthritis	—	Marked
2	+	1	140	Canary yellow	10	++	0	3 3 3 3 2 1 1 — — —	Cord Compression and syphilis ?	—	Marked
3	+	4	150	Golden tint	8	+	+	5 5 5 4 4 3 2 1 — —	Tabes	+	Marked
4	+	1	130	Golden tint	0	0	±	3 3 3 2 2 1 1 — — —	?Tabes ?	—	Slight
5	—	No treatment	30	Greenish yellow	4	+	0	— — — — — — — — — —	Carcinoma of pancreas	—	Moderate
6	—	6	140	Golden	0	0	0	— — — — — — — — — —	Syphilis	—	Moderate

* In all the cases the surface tension was normal; nitrous acid, negative; iodin, negative; bile tests on urine, positive.

DISCUSSION

DR. H. C. SOLOMON: The frequency with which Dr. Mella found coloration in the spinal fluid is much greater than the average. We have performed puncture in a number of patients with arsphenamin jaundice and rarely have found coloration of the fluid. Recently, I performed a necropsy in a case of acute yellow atrophy. All the peritoneal fluid and practically all the organs were colored, but in the central nervous system there was not the slightest trace of color. On the other hand, I have performed necropsy in cases of acute jaundice in which the coloration was very marked in the central nervous system. Gennerich states that there is a coloration which comes only when concentration of the bile pigments in the blood reaches a certain point, then the central nervous system will be colored and the color will remain there longer than anywhere else in the body. Schmorl states that, in three cases in which he found yellow fluid in the ventricles, there had been some injury of the choroid plexus and the fluid came through in large amounts into the ventricles.

DR. HUGO MELLA, closing: Regarding the color of the fluids, I have always asked some disinterested person to observe the color so as to check up on its presence. With the quick response to bile tests on the urine and the negative responses in the spinal fluid, we have not proved the presence of bile in the fluid in these jaundice cases, nor have we been able to determine the cause of xanthochromia.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, April 28, 1922

C. H. FRAZIER, M.D., *President*

ROENTGEN-RAY LOCALIZATION OF A GLIOMATOUS CYST BY THE INJECTION OF AIR. DR. F. C. GRANT.

The patient presented symptoms and physical signs which suggested the diagnosis of brain tumor in the right motor area. At operation, reflection of the bone flap revealed an extremely tense dura. The pressure was reduced sufficiently, by callosal puncture and by intravenous injection of a 15 per cent. salt solution, to permit opening the dura in sections. As one section was opened an area of the cortex which felt cystic was encountered. A needle puncture evacuated 25 c.c. of fluid, and air was injected into the cyst cavity. Roentgen-ray studies localized the cyst accurately and showed a filling defect in its wall which later proved to be a gliomatous mass. A second operation was performed a week after the first. The osteoplastic flap was again laid back, the dura freely opened and the cortex incised, revealing the cyst. A nubbin of fairly well encapsulated gliomatous tissue about 3 cm. in diameter was removed. The cyst wall was too delicate to permit clean removal. An uneventful postoperative recovery ensued with beginning return of function on the left side. After a thorough course of radium treatment the patient was discharged.

CLINICAL RESULTS WITH MORE EXACT TECHNIC FOR ALCOHOLIC INJECTION OF THE SECOND AND THIRD DIVISIONS OF THE TRIGEMINAL NERVE. DR. F. C. GRANT.

The more exact technic for alcoholic injection of the second and third divisions of the trigeminal nerve by using the zygometer and measuring the horizontal and vertical angles between the needle shaft and the skin of the cheek with a protractor was outlined. Three types of cases were treated—major trigeminal neuralgia, malignant growths situated in the distribution of the fifth nerve and a case of masseter spasm. The conclusions were:

1. Alcohol injections of the three divisions of the trigeminal nerve are of value in treatment of tic douloureux, and as an adjunct to the treatment of painful growths about the face, tongue and jaws and in masseter spasm.

2. By the use of the zygometer and protractor an attempt has been made to render more accurate the description of the technic for injecting the maxillary and mandibular divisions of these nerves.

3. Clinical experience has coincided with anatomic studies. Injections of the maxillary division should be made from the 3 cm. mark, the needle should subtend an angle of 100 degrees from above downward in the horizontal plane, and 115 degrees from before backward in the vertical plane. The nerve is reached from 5 to 5.5 cm. from the surface.

4. Injections of the mandibular division should be made from the 2 cm. mark, the needle should subtend an angle of 90 degrees in the horizontal plane and 110 degrees from above downward in the vertical plane. The nerve is reached from 4.5 to 5 cm. from the surface.

5. By applying these facts to a surface of injections clinically, the percentage of failures to reach the nerve trunks has been materially reduced.

A CASE OF ENCEPHALITIS EXHIBITING HYPERTHYROID SYMPTOMS AND LATER THE PARALYSIS AGITANS SYNDROME.

Dr. F. H. Leavitt.

This case is of interest in that the patient gave a history of a febrile condition, which was undoubtedly an attack of epidemic encephalitis. Some months later he exhibited a clinical picture of acute hyperthyroidism and was treated for this condition in the hospital and dispensary. A few months following this, these symptoms were gradually replaced by the picture of a paralysis agitans syndrome, which he still shows. The disease has steadily but slowly progressed, and the only medication which has afforded any relief is digitalis in large doses.

DISCUSSION

Dr. Charles S. Potts: So far as my experience goes, a rapid pulse in encephalitis, which this patient had, is not uncommon. I have seen several patients in whom the temperature was practically normal, but the pulse rate varied from 110 to 130. All the other symptoms of epidemic encephalitis were present in this case.

Dr. C. W. Burr: The patient is probably suffering from a sequela of encephalitis, and the striking thing in his case is that on his first admission to the hospital the condition much resembled hyperthyroidism. When he came back the second time the condition resembled paralysis agitans.

Dr. Leavitt, in closing: We consider the case one of encephalitis, but the history given antedated the time when he showed hyperthyroidism, so that the encephalitis which may have occurred did not occur between the time he had hyperthyroidism and paralysis agitans. That was the confusing part.

LESIONS OF THE OPTIC CHIASM AND TRACTS WITH RELATION TO THE MIDDLE CEREBRAL ARTERY WITH REPORT OF CASE.

Dr. Temple Fay.

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MANGANESE POISONING: REPORT OF A CASE WITH SYMPTOMS SUGGESTIVE OF BILATERAL LENTICULAR INVOLVEMENT.

Dr. George Wilson.

Manganese poisoning was first described in 1834; the first detailed account, however, was not published until von Jaksch wrote his first article, in 1901; Edsall and Drinker and Casamajor of this country have also written on the subject. No pathologic changes have been found in the nervous system in the few cases which have come to necropsy, although no mention is made of a careful investigation of the basal ganglions. A considerable degree of biliary cirrhosis of the liver has been found, and the liver cells contain a great deal of pigment.

After from six months to three years of work in separating manganese from zinc, men may show symptoms of poisoning, the first being a disturbance in walking down hill. Men who work with manganese frequently notice this symptom, and they call the condition being "zinced." For instance, a man who wheels a barrow down an incline, often in the early stage of manganese poisoning, falls forward, a condition that might be called propulsion. Retropulsion and lateropulsion may also occur. Von Jaksch described a gait in which the

men walked on their metatarsophalangeal joints. Another peculiarity in the gait is that walking backward is impossible. Speech is slow, monotonous and sometimes slurring; Edsall calls it an "economical speech." The facies are marked and attacks of involuntary emotionalism may occur.

Animal experimentation has produced no results. The method of production of manganese poisoning seems to be through the inhalation of dust; men who work in the so-called water process in separating manganese do not develop symptoms, whereas those who work in the dust do.

The case which I report is that of a man, aged 31 years, whom I examined at the U. S. Veterans' Bureau on April 13, 1922. In October, 1917, he was struck by 13,000 volts of electricity and was unconscious for two hours. He denies venereal disease. He has been married four years and has one child living and well. He worked in manganese for four years prior to 1918.

A few weeks before his induction into the army on April 2, 1918, he began to have trouble in walking. This difficulty has gradually progressed, although it differs in severity from time to time. He says that his arms draw up; by this he means that they go into a condition of spasm. Without feeling at all depressed he sometimes has crying spells. While he had symptoms before he was inducted into the service, they were intensified by three weeks of drilling. He fell on the drill ground and was unconscious for one hour, and since that time his symptoms have progressed rapidly. He has difficulty in bringing the hands to the face, especially on the right side. An extraordinary symptom about which this man complains occurs during sexual intercourse. When copulation is attempted connection is made with facility, but at that point a halt occurs, and the man, because of a condition which may be perseveration of certain groups of muscles, can neither push forward nor retreat until some minutes have elapsed and the spasm disappears.

Physical Examination.—The face was flushed and masked, and the face and body in general were bathed in perspiration. The station, pupils, cranial nerves, deep and superficial reflexes and sensation were all normal, as were also the various laboratory examinations. Speech was low toned and monotonous. The gait was the most striking thing in this man's case. He walked with the legs markedly extended and rather widely separated, dragging the toes along the floor, and because of a tendency to propulsion it seemed as though each step would end in a fall. At times, though rarely, he showed lateropulsion. When the man attempted to walk backward, he fell. He had difficulty in sitting down and in turning over in bed. The legs sometimes became extremely rigid with intense contraction of the thigh muscles. The right upper extremity frequently assumed a forced attitude, the arm being abducted and the elbow and fingers flexed. In the finger to nose test there was difficulty in reaching the goal, especially on the right, because during action the muscles became rigid, which prevented coordinated movement.

This man presented signs that to me are indicative of organic disease, most likely of the basal ganglions—the facies, the speech, the rigidity and vasomotor disturbances all bespeak anatomic change, although some probably would conclude that because the man was seeking compensation the case must be one of hysteria.

DISCUSSION

DR. CHARLES K. MILLS: Possibly this patient may have had some hysterical symptoms or tendency as some patients with organic cases have, but I do not believe the case is one of hysteria. He had four or five symptoms which seem

to point to organic and probably lenticular disease; these are the forced attitude of arm and leg which are much like those of a case of degeneration of both striate bodies reported by me, distortion on movement, monotonous speech and some points in his history. I am fully in accord with those who criticize the too frequent diagnosis of hysteria. During many years of neurologic practice, I have observed that one mistake most frequently made by men whom we all regard as competent, is that of diagnosing hysteria when organic disease is present. Among the diseases regarding which this mistake is most frequently made are disseminated sclerosis, myasthenia gravis, brain tumor, spinal tumor, lenticular degeneration, localized encephalitis, combined sclerosis, and many other affections.

Dr. Charles W. Burr: This man gave Dr. Wilson a history unlike the one he gave to me. To me he said that he had been perfectly well until he received the electric shock. The gait was not exactly as it is at the present time, and my recollection is that it was as bad in one leg as it was in the other. He was a patient of mine at the Orthopedic Hospital and he became much better, so that he walked about the wards, perfectly well; then one day he received a letter from his wife; there was a row and he refused to stay in the hospital and went out. He walked without any help, walked with a perfectly natural gait, although somewhat slowly and weakly, and his speech became absolutely normal. That was, I think, about two years ago. He then gave me the history of electric shock and dated all his troubles from that. In my opinion this is a case of hysteria.

Dr. J. Hendrie Lloyd: Has anybody ever seen a case of manganese poisoning like this before? To me, it is like nothing but hysteria. The man, I have a strong suspicion, has a psychoneurosis. I do not see how Dr. Wilson can make out lenticular degeneration in this case. These bizarre disorders are usually psychotic. This man never hurts himself; he is emotional; he had a brief service in the army, which did not agree with him; he had to leave the army in three weeks; and he has been a claimant ever since. That settles the diagnosis for me.

Dr. T. H. Weisenburg: According to the history, this patient had symptoms four months prior to his induction into the service. This is against the theory that the patient was hysterical to avoid service. The gait of the patient, while bizarre, nevertheless is not unlike that of a patient who had been in the wards for nervous diseases of the Philadelphia Hospital for many years. This patient's case had been variously diagnosed—at one time as hysteria, then chorea and now as a striate case. In the course of years many cases have been presented before this society in which diagnoses of hysteria have been made and which have turned out to be organic cases of various types. I agree entirely with Dr. Wilson that this case is organic, and that probably a striate lesion is present.

Dr. Williams B. Cadwalader: Manganese is regarded as a general systemic poison. So far as I know, a postmortem study has never been made on such a case that showed lesions affecting the basal ganglions of the brain. It is hard to believe that the lenticular nuclei can be affected. In certain smelting processes, however, it is known that workmen may be poisoned by carbon monoxid. Furthermore, carbon monoxid may have a selective action on the blood vessels of the basal ganglions, and in this way bilateral softening of the lenticular nuclei may be produced. Drs. McConnell and Spiller have reported a case of illuminating gas poisoning before this Society in which they found

lesions of both lenticular nuclei at postmortem. During the lifetime of their patient there had been evidences indicating such lesions. I offer this as a suggestion to explain the symptoms in Dr. Wilson's patient. The first case of true progressive lenticular degeneration exhibited before this society was presented by me in 1912 and is recorded in the proceedings. At that time the opinion was expressed by some that it resembled hysteria. The differential diagnosis may be confusing. Notwithstanding the evidence presented by Dr. Wilson, I do not feel that his patient presents characteristics of true organic cerebral disease; the case impresses me as being one of pure hysteria.

Dr. F. X. Dercum: Has this man been observed when he himself has not known he was under observation? I think it is most important to know how this man conducts himself when he is alone; and how he conducts himself when food is placed on the table, whether he has the same difficulty in bringing food to his mouth as in bringing his hand to nose. We are sometimes forced to follow such a plan when we suspect malingering, and I think this is eminently the kind of case in which such a procedure should be followed.

Dr. N. W. Winkelman: There is a tendency to assume that because a man has been in the service and is seeking compensation, he is either a malingerer or a neurotic. This is a mistake. I have in mind a case studied in on of the hospitals here and diagnosed hysteria. At operation a tumor of the cord was found. The patient died, but his death was due in large part to the delay in operation. I agree with Dr. Wilson's diagnosis.

Dr. Wilson, in closing: I agree with those who say the gait of this man is bizarre; but is not the gait of certain patients with organic disease often bizarre? Take the gait in far advanced tabes or dystonia musculorn deformans or the festinating gait of paralysis agitans—are they not bizarre? The fact that the patient improved, as stated by Dr. Burr, is not proof so much that the patient necessarily was suffering from hysteria as it is that Dr. Burr knows how to employ psychotherapy. Real psychotherapy should not only cure the patient with the functional case, but it should make the patient with the organic case think that he is well, and this is what Dr. Burr did for his patient. I fully agree with Dr. Lloyd that this man's army experience did not agree with him—what man with a case of lenticular disease ever did enjoy military service? I cannot do more than reiterate the caution which Dr. Winkelman has expressed, namely, that if we assume that a man, because he is a claimant for compensation, is a malingerer or has hysteria, we will make grave diagnostic errors. So far as I know, carbon monoxid does not enter into the symptomatology of manganese poisoning. Dr. Cadwalader's remarks that the first case of progressive lenticular degeneration presented to this society was thought by some to be hysteria, are apropos. History is repeating itself, for while this patient does not have Wilson's disease, I believe that he has lenticular degeneration.

TRISMUS DEVELOPING IN THE COURSE OF A TUMOR OF THE PONS. Dr. George Wilson.

The following case is presented because of the development of trismus which came on in a man suffering from a tumor of the pons. This has been reported before, but is, I believe, sufficiently rare to warrant presentation.

A white man, 44 years of age, was admitted to the Episcopal Hospital on Jan. 20, 1922. His family and past histories were negative. His chief complaint

was double vision and dizziness. Last September he first noticed diplopia when looking at an object to one side. Two weeks later he began to have nausea and vomiting, although the vomiting was not projectile. He had had no headaches since the onset, but he said that his head felt numb all the time. For the last two weeks he has been unable to open his mouth completely, and he becomes dizzy after he sits up.

Physical Examination.—In the Romberg position there was marked swaying with the eyes closed, and the man immediately became dizzy, nauseated and vomited. The left seventh nerve showed weakness, especially in the lower branch. The left masseter was distinctly weak and on opening the mouth the jaw deviated to the left. The jaw could be opened only a short distance, and this condition of trismus gradually became worse during the man's stay in the hospital. The lower jaw could not be pushed down with moderate force. The tongue was protruded in the midline and was not atrophic. Neither eyeball could be moved outward or inward; the left eyeball moved slightly inward on attempts at convergence; upward and downward movements were well performed. Bilateral corneal anesthesia was present. The left palpebral fissure was narrower than the right. The eyegrounds were normal.

Asynergy and astereognosis were easily demonstrated in the right hand. Pain sense was diminished on the right side of the face, in the right arm and hand. All the deep reflexes were present and active, the right patellar reflex being more active than the left. Plantar stimulation produced flexion on both sides.

Smell and taste were normal. The ears were examined by Dr. Watson, who reported 1/18 hearing in the left ear and 2/18 in the right ear. The various laboratory reports were negative.

Book Reviews

THE DEFECTIVE, DELINQUENT AND INSANE. The Relation of Focal Infections to Their Causation, Treatment and Prevention. By HENRY A. COTTON, M.D., Medical Director, New Jersey State Hospital at Trenton. Pp. 192. Princeton: Princeton University Press, 1921.

It is fortunate that Dr. Cotton has condensed all of his views into one book. For a number of years, his theories have been given wide publicity, not only in the medical but also in the lay press. His views, with pictures of the author, the hospital, operating room, etc., have been syndicated throughout the country in the magazine sections of Sunday newspapers and in a number of magazines, including the *Literary Digest*. It is not in the domain of the reviewer to question the means by which such publicity has been gained, but it is only fair to call attention to it.

In brief, Dr. Cotton divides insanity into two kinds: (1) That form which is the result of definite organic changes, such as are brought about in paresis by the spirochete; (2) the functional psychoses—which term he rejects—which, according to him, are due to disorder of the brain arising from "a combination of many factors, some of which may be absent, but the most constant one is an intracerebral, biochemical, cellular disturbance arising from circulating toxins, originating in chronic focal infections situated anywhere throughout the body and probably to some extent in disturbances of the endocrine system." In this group he includes the manic-depressive psychoses, dementia praecox and paranoic conditions or chronic delusional states. He rejects what he calls the "old idea" that these mental disorders are in any way the result of constitutional defects. He takes issue with the freudians who believe that psychoses may have a sexual basis.

His method of handling this so-called toxic group consists, first, in making a diagnostic survey, which differs in no way from that used by any physician or well regulated hospital. It is in the interpretation of the results that Dr. Cotton differs from many physicians. According to him, most infection lies, first, in the teeth; second, in the tonsils; then in the gastro-intestinal tract, genital organs, sinuses, etc. After removal of teeth, tonsils or any organ cultures are made and autogenous vaccines are given.

To quote from Dr. Cotton's book, page 107: "Every patient should receive a course of treatment by autogenous vaccines after the infected teeth and tonsils are removed but not before because of the probability of a severe reaction. Within a week or two after the vaccine treatment has been completed, a course of treatment by anti-streptococcus and colon bacillus sera should be given. By these two methods the systemic infection should be eradicated. If the patient fails to recover and further examination reveals severe intestinal infection, then operation for removal of the infected area of the colon should be the next step. If the cervix, or the seminal vesicles are found to be infected they should also be removed by surgical means. No ill effects have been noted from the administration of either vaccines or serums."

Every tooth which is under the slightest suspicion is promptly removed. In fact Dr. Cotton prefers to err on the side of removal, rather than to save the tooth. The tonsils are treated with less consideration for practically all

of them are removed. The author has no hesitancy in advising resection of the colon, for he mentions that 150 of these operations have been performed for him by Dr. John W. Draper. In the female, operations on the genital organs, and in the male, on the seminal vesicles, are not at all uncommon.

Dr. Cotton reports a number of illustrative cases and compares the results obtained in the hospital in the last three years, during which time the present method of treatment has been in vogue, with that of former years, and he states that the residence of the patients has been less and that there are fewer chronic cases. For example, in 1918, there were admitted to the New Jersey State Hospital 380 patients, classified in the so-called functional toxic group. At the end of the year there remained 160, and at the end of the second year. sixty patients were in the hospital. Of these, twenty returned, and twelve of that number were found to have minor infections which had been overlooked. The remaining eight were found to have serious intestinal involvement and later were operated on. Of these, only four are in the hospital at the present time. Two and a half years later only fifty-one of the original 380 were in the hospital, and of these nine were convicts.

The author then emphasizes that readmissions to the hospital are not larger than the average for a ten year period before 1918. He states that the length of time in which patients recovered spontaneously prior to this method of treatment was ten months, whereas in the last three years it has been only three months. He is careful to state that good results are not obtainable in the old cases of dementia praecox and can be expected only in the cases in which treatment is instituted early. In the next to the last chapter of his book, he discusses the defective types; he says that there are a great many in whom similar toxic disturbances are causative factors, and if these were removed, the patients would recover.

The foregoing statements are made so as to give the reader an adequate idea of the writer's point of view. It is, of course, difficult to find fault with a method of treatment in which infections are removed. The majority of surgeons, however, will disagree with Dr. Cotton in his advocacy of resection of the colon and of the seminal vesicles. Perhaps many internists will differ with his routine method of vaccine treatment. A preponderant number of competent dentists do not agree with his point of view. But what is there in Dr. Cotton's theory of the removal of real focal infections which varies from that held by most physicians?

Until a few years ago it is true, as Dr. Cotton has stated, that most institutions for the insane were content to do nothing for their patients; they were simply large boarding houses. Unfortunately, this is still the case, not only in this country but throughout the entire world, and Dr. Cotton deserves a great deal of praise for his "do something" policy, especially in consideration of its having stirred to action the authorities at a great many institutions who are beginning to scrutinize their patients. On the other hand, must he indiscriminately remove teeth, tonsils and internal organs because he has a theory to uphold?

The reviewer, as well as other neurologists of Philadelphia, are qualified to discuss Dr. Cotton's work, because they have seen some of his results. Quite a large number of Dr. Cotton's "cured" patients have fallen into their hands. It is possible that Dr. Cotton, in defending his point of view, would say that all focal infection had not been removed. The chiropractor offers a somewhat similar apology for his failures in that the patient is told that he has not had sufficient adjustment.

Dr. Cotton's statistics of admissions and discharges and the so-called cures in the New Jersey State Hospital at Trenton are in no way better than those of any up-to-date institutions, in spite of the fact that the latter practice conservative methods and continue in the belief that dementia praecox has hereditary and constitutional factors in its causation.

In medicine, as well as in everything in life, it is never wise to be too dogmatic, for there is always a possibility that one's point of view may be wrong. Dr. Cotton, however, has burned his bridges behind him, for on page 122 he expresses sorrow for "these afflicted individuals" who differ from him.

In the meantime the removal of teeth, tonsils, intestines and seminal vesicles will be continued.

ANISOCORIA ON LOOKING LATERALLY (THE REACTION OF TOURNAY). By Dr. André Noyer. Pp. 90. Lescuyer Frères, 1921.

In a monograph of ninety pages Noyer gives the results of his investigations of a reaction described by Tournay in 1917 and 1918. This reaction consists of the production, on looking laterally, of an inequality of the pupils which is due to dilatation of the pupil of the abducted eye and appears after the eyes have been carried laterally and held there for a few seconds. The inequality was shown by Noyer to be due to dilatation in the abducted eye and not to contraction of the adducted eye. The reaction is independent of the stimulus of light and can be demonstrated in a dark room by means of the light reflected from an ophthalmoscopic mirror.

Noyer examined this reaction in several thousand normal people and found it present in all of them. It is much easier to obtain in those with moderately large pupils than in those with small. Emmetropia, hypermetropia, myopia and astigmatism do not interfere with the reaction. The average increase in the size of the pupil as measured by special apparatus is 0.5 mm.

In an experiment to determine the presence of the reaction in various animals, Noyer established the following:

1. Animals having eyes comparable in location and mobility to those of man have a positive Tournay reaction (cat and dog).

2. Animals having lateral and mobile eyes have a negative Tournay reaction (horse).

3. Animals having eyes in an extremely lateral position, almost immobile and with very dilated pupils (rabbit) show a permanent Tournay reaction in each eye.

Cocain, atropin and pilocarpin prevent the reaction from occurring. Epinephrin prevents it from occurring in those segments of the pupil which are dilated.

Noyer tested the reaction in a large number of cases showing various diseases and reports the results in 386 of them. In disorders of the eye in which the pupil remained mobile, the reaction was obtained in spite of the lack of perception of light due to cataract or to optic atrophy. This indicated that the reaction was not essentially a sensorimotor reflex but an associated movement completely independent of vision. In diseases of the sympathetic supply to the iris the reaction was normal. It is also normal in general conditions, such as syphilis, exophthalmic goiter, rheumatism and pneumonia. In dis-

eases of the nervous system the reaction was absent in only 15 cases: 7 of general paralysis, 1 of tabes, 3 of multiple sclerosis, 1 of poliomyelitis and 2 of Parkinson's disease.

Noyer concludes by stating that the number of cases examined was too small to warrant definite conclusions, but he believes that the reaction may possibly come to have a value analogous to that of the Argyll Robertson pupil.